University of Alberta

Strength, muscle morphology, myosin isoforms and cortisol responses following resistance training with manipulation of concentric and eccentric muscle actions in recreational resistance-trained women.

by

Ellen Maureen Gillies ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Faculty of Physical Education and Recreation

Edmonton, Alberta
Spring 2004
University of Alberta

Library and
Archives Canada

Published Heritage
Branch

395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque et
Archives Canada

Direction du
Patrimoine de l'édition

395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file Votre référence
ISBN: 0-612-96271-7
Our file Notre référence
ISBN: 0-612-96271-7

The author has granted a non-exclusive license allowing the Library and Archives Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque et Archives Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

L'auteur conserve la propriété du droit d'auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou aturement reproduits sans son autorisation.

In compliance with the Canadian Privacy Act some supporting forms may have been removed from this thesis.

Conformément à la loi canadienne sur la protection de la vie privée, quelques formulaires secondaires ont été enlevés de cette thèse.

While these forms may be included in the document page count, their removal does not represent any loss of content from the thesis.

Bien que ces formulaires aient inclus dans la pagination, il n'y aura aucun contenu manquant.

Canadä

ABSTRACT

The objective of this study was to examine changes in dynamic strength, muscle fibre cross-sectional area, myosin heavy chain (MHC) composition and cortisol following resistance training with manipulation of the time to complete the concentric (CON) and eccentric (ECC) muscle actions. Twenty-eight women [mean age - 24.3 (SD 5.8) yr, height - 164.6 (7.3) cm, body mass - 66.5 (9.2) kg, body fat – 17.4 (5.0)%] completed 3 training sessions a week for 9 weeks. Two sets of 4 lower body exercises (incline leg press, parallel squat, leg extension, leg flexion) were completed during each session using a 6-8 RM intensity. The Long Concentric (LC) group performed the CON action in 6 s and the ECC action in 2 s, while the Long Eccentric (LE) group completed the CON and ECC phases in 2 and 6 s, respectively. Both groups experienced similar increases in leg press CON, ECC and combined ECC/CON maximal strength ($P \leq 0.01$). The increase in ECC 1 RM (44%) was greater ($P \leq 0.01$) than the increases in ECC/CON (25%) and CON (21%) 1 RM for both groups. Immunohistochemical analyses using MHC isoform specific monoclonal antibodies demonstrated that both type-I (16%) and type-IIA (26%) muscle cross-sectional areas of the vastus lateralis increased ($P \leq 0.05$) following LC training while only type-I (11%) fibres increased ($P \leq 0.05$) following LE training. MHC composition was altered in both groups, with a decrease in MHCIId(x) and a concomitant increase in MHCIIa ($P \leq 0.05$). Twenty-four-hr urinary cortisol increased ($P \leq 0.05$) in LC but not LE following training. These data show that emphasis on either the CON or ECC muscle action are both effective for enhancing strength in healthy adult women. Additionally, preferential hypertrophy can be induced with manipulation of CON and ECC muscle action time, with

similar alterations in MHC composition. These findings were not attributable to a differential response to training volume, but to the manipulation of the duration of the CON or ECC action. Furthermore, there was some suggestion that the adaptations noted were due to greater metabolic involvement with LC.

ACKNOWLEDGEMENTS

I would like to thank my advisor Dr. Gordon Bell for his support, enthusiasm and mentorship.

A sincere thanks to my committee members, Dr. Ted Putman and Dr. Dan Syrotuik for their expertise, comments and always being available. I would like to express my appreciation to my examining committee members, Dr. V. Harber and Dr. M. Haykowsky for their time, comments and suggestions. A special thanks to Dr. P. Chilibeck of the University of Saskatchewan for his function as external examiner. As well, I would like to thank Dr. Tom Martin for performing the muscle biopsy procedure and Dr. Paul Zehr for his involvement with my first project.

I would also like to acknowledge the assistance with laboratory procedures provided by Ian MacLean, Jean Pearcey, Jeremy Bamford and Alex Lay.

Finally, I would like to thank all of the subjects that participated in my projects for their time and effort. I would especially like to thank the women of the resistance training study that unhesitatingly and enthusiastically did all that was asked of them.

TABLE OF CONTENTS

CHAPTER I - REVIEW OF LITERATURE 1
INTRODUCTION 1
Differences between Concentric and Eccentric Muscle Actions *1*
Maximal strength 1
Mechanical 2
Metabolic 2
Neural 3
Muscle Damage/Injury following Eccentric and Concentric Muscle Actions *6*
Ultrastructural indication of muscle damage 6
Prior bout of eccentric exercise and muscle damage with continued resistance training 6
Markers of muscle damage 7
Estrogen and Muscle Cell Membrane Integrity *7*
Exosarcomeric Muscle Proteins *9*
Theories Regarding Mechanisms of Muscle Hypertrophy with Resistance Training *10*
Cortisol Action on Skeletal Muscle Tissue *12*
Strength and Hypertrophy Responses with Concentric and Eccentric Muscle Actions *13*
Strength following DCER training with equal loads for concentric and eccentric actions 14
Hypertrophy and alterations in MHC isoforms following DCER training with equal concentric and eccentric loads 15
Strength following DCER training with a greater load for eccentric actions 16
Hypertrophy following DCER training with a greater load for eccentric actions 17
Conclusions regarding concentric and eccentric muscle actions during DCER training 18
CHAPTER II - STRENGTH, MUSCLE MORPHOLOGY, MYOSIN ISOFORMS AND CORTISOL RESPONSES FOLLOWING RESISTANCE TRAINING WITH MANIPULATION OF CONCENTRIC AND ECCENTRIC MUSCLE ACTIONS IN RECREATIONAL RESISTANCE-TRAINED WOMEN. 19
INTRODUCTION 19
PURPOSE OF THE STUDY 20
STATEMENT OF PROBLEM AND SIGNIFICANCE OF STUDY 20
HYPOTHESES 22
LIMITATIONS 23
DELIMITATIONS 23
METHODS 24
Subjects *24*
Experimental Design *24*
Resistance Training Protocols *25*
Strength Assessment *27*
Relative Training Load (% 1RM) *28*
Weekly Repetitions per Set *28*

Weekly Training Load *28*
Training Volume *29*
Muscle Sampling *29*
Immunohistochemistry for Myosin Fibre Typing *29*
Morphometric Analysis *31*
Muscle Protein Preparation *31*
Electrophoretic Analysis of MHC Isoforms *32*
Cortisol *33*
Dietary Monitoring *34*
STATISTICAL ANALYSIS 34
RESULTS 35
Subjects *35*
Training Compliance *35*
Strength Assessment *36*
Leg Press 36
Bench Press 36
Training Variables *37*
Leg Press 37
Squat 38
Leg Extension 38
Total Training Volume 39
Fibre Type Cross-Sectional Areas and Proportions *40*
MHC Isoform Analyses *41*
Cortisol *42*
Dietary Intake *43*
DISCUSSION 43

CHAPTER III GENERAL DISCUSSION AND CONCLUSIONS 78

REFERENCES 84

APPENDIX A MUSCLE ACTIVATION DURING ECCENTRIC AND CONCENTRIC MUSCLE ACTIONS OF VARYING DURATION WITH A SUBMAXIMAL LOAD 97

INTRODUCTION 97
METHODS 100
Subjects *100*
Experimental Setup *100*
Experimental Procedures *101*
Data Collection *103*
Data Analysis *104*
Data Reduction *104*
Statistical Analysis *105*
RESULTS 106
Velocity and Acceleration *106*
Peak Velocity *108*
Kinetic Pattern *108*
Average EMG *109*

Average EMG of Bins *110*
Peak EMG *111*
Temporal Pattern of Peak EMG *112*
Muscle Activity On/Offset *113*
Pre/Post Movement Muscle Activity *114*
DISCUSSION 114

REFERENCES **121**

APPENDIX B DIETARY INTAKE RECORDS FOR ALL RECORDING PERIODS **135**

APPENDIX C **137**

PARTICIPANT INFORMATION LETTER AND INFORMED CONSENT **137**

LIST OF TABLES

TABLE 1. EXPERIMENTAL DESIGN AND ASSESSMENT SCHEDULE. 63
TABLE 2. ANTHROPOMETRIC MEASUREMENTS AND BODY COMPOSITION PRE- AND POST-TRAINING. 64
TABLE 3. LEG PRESS MAXIMUM STRENGTH (1 RM) FOR THE THREE STRENGTH TESTS AT PRE-, MID- AND POST-TRAINING. 65
TABLE 4. BENCH PRESS MAXIMUM STRENGTH (1 RM) F OR THE THREE STRENGTH TESTS AT PRE-, MID- AND POST-TRAINING. 66
TABLE 5. RELATIVE TRAINING LOAD (PERCENTAGE OF MAXIMAL COMBINED 1 RM LEG PRESS STRENGTH) DURING WEEKS 1, 5 AND 9. 67
TABLE 6. TRAINING VARIABLES OF WEEKLY MEAN REPETITIONS PER SET, MEAN LOAD PER SET AND TOTAL VOLUME (SUM OF REPETITIONS X LOAD FOR EACH SET) FOR LEG PRESS THROUGHOUT THE 9 TRAINING WEEKS. 68
TABLE 7. TRAINING VARIABLES OF WEEKLY MEAN REPETITIONS PER SET, MEAN LOAD PER SET AND TOTAL VOLUME (SUM OF REPETITIONS X LOAD FOR EACH SET) FOR SQUAT EXERCISE THROUGHOUT THE 9 TRAINING WEEKS. 69
TABLE 8. TRAINING VARIABLES OF WEEKLY MEAN REPETITIONS PER SET, MEAN LOAD PER SET AND TOTAL VOLUME (SUM OF REPETITIONS X LOAD FOR EACH SET) FOR LEG EXTENSION THROUGHOUT THE 9 TRAINING WEEKS. 70
TABLE 9. TOTAL TRAINING VOLUME (WEEKLY SUM OF REPETITIONS X LOAD FOR EACH SET OF LEG PRESS, SQUAT AND LEG EXTENSION) THROUGHOUT THE 9 TRAINING WEEKS. 71
TABLE 10. MUSCLE FIBRE CROSS-SECTIONAL AREA (μM^2) PRE- AND POST-TRAINING. 72
TABLE 11. MUSCLE FIBRE TYPE PROPORTIONS (%) PRE- AND POST-TRAINING. 73
TABLE 12. MYOSIN HEAVY CHAIN COMPOSITION (% OF TOTAL MHC) PRE- AND POST-TRAINING. 74
TABLE 13. TWENTY-FOUR HOUR URINARY CORTISOL (μG PER 24 HOURS) DURING WEEKS 1 AND 9. 75
TABLE B-1. MEAN DAILY CALORIC INTAKE AND MACRONUTRIENT INTAKE (% OF DAILY CALORIC INTAKE) OF A 3-4 DAY PERIOD MEASURED EVERY 2 WEEKS THROUGHOUT THE 9 WEEKS OF TRAINING. 136

LIST OF FIGURES

FIGURE 1. REPRESENTATIVE MHC IMMUNOHISTOCHEMICAL STAINS OF SERIAL CROSS-SECTIONS OF THE VASTUS LATERALIS MUSCLE. 76
FIGURE 2. REPRESENTATIVE GELS SHOWING MHC ISOFORMS SEPARATED BY SDS-PAGE. .77
FIGURE A-1. EXPERIMENTAL SETUP. 124
FIGURE A-2. SAMPLE DATA FOR A SINGLE SUBJECT PERFORMING CONCENTRIC AND ECCENTRIC CONTRACTIONS OF THE TRICEPS SURAE MUSCLES AT THE SLOWEST PERFORMANCE VELOCITY (V1). 125
FIGURE A-3. KINEMATIC DATA OF VELOCITY (A) AND ACCELERATION (B) DURING THE 3 VELOCITY CONDITIONS. 126
FIGURE A-4. AVERAGE BIN FORCE DURING CONCENTRIC AND ECCENTRIC MOVEMENTS AT ALL VELOCITIES. 127
FIGURE A-5. RELATIONSHIP BETWEEN NORMALIZED VELOCITY AND AVERAGE EMG FOR BOTH MUSCLE ACTION CONDITIONS AND ACROSS ALL MUSCLES. 128
FIGURE A-6. AVERAGE EMG DURING BOTH MUSCLE ACTIONS AND ACROSS ALL MUSCLES. 129
FIGURE A-7. NORMALIZED AVERAGE EMG FOR EACH MOVEMENT BIN FOR ALL MUSCLES DURING CONCENTRIC (CON) AND ECCENTRIC (ECC) MUSCLE CONTRACTIONS. 130
FIGURE A-8. PEAK EMG OF ALL MUSCLES DURING CONCENTRIC AND ECCENTRIC MUSCLE ACTIONS AT ALL VELOCITIES. 131
FIGURE A-9. TIME TO PEAK EMG NORMALIZED TO MOVEMENT TIME FOR ALL VELOCITY AND MUSCLE ACTION CONDITIONS. 132
FIGURE A-10. MUSCLE ACTIVITY ON/OFFSET. 133
FIGURE A-11. PRE/POST-MOVEMENT MUSCLE ACTIVATION. 134

LIST OF ABBREVIATIONS

3-MH – 3-methylhistidine

AB - antibody

aEMG – average electromyography

CV – coefficient of variation

CON – concentric

DCER – dynamic constant external resistance

ECC – eccentric

EMG - electromyography

hGH – human growth hormone

IF – intermediate filament

LG – lateral gastrocnemius

LC - long concentric muscle action

LE - long eccentric muscle action

MG – medial gastrocnemius

mAb – monoclonal antibody

MHC – myosin heavy chain

MVC -maximal voluntary contraction

pEMG – peak electromyography

ROM – range of movement

RM – repetition maximum – the maximal number of repetitions that a resistance (load/weight) can be lowered and lifted using correct technique and adherence to the specified tempo

SSC – stretch-shortening cycle

SOL – soleus

TUT – time under tension or the time to complete a muscle action(s)

TA – tibialis anterior

VL – vastus lateralis

CHAPTER I - REVIEW OF LITERATURE

Introduction

Skeletal muscle is a dynamic tissue that responds to alterations in functional demands. Tension is produced by muscle as it shortens or lengthens. Asmussen (1953) first coined the terms concentric and 'excentric' (eccentric) to describe dynamic muscle action. There are known differences between these two actions in terms of tension generation (Katz, 1939), neural activation (Bigland & Lippold, 1954) and energy cost (Asmussen, 1953). Despite the long history of known differences between concentric and eccentric muscle actions, the role of these actions in influencing adaptations to resistance exercise is not yet completely understood. This brief review will highlight some of the differences between concentric and eccentric actions, identify a few areas of current research, provide an overview of theories regarding muscle hypertrophy, examine cortisol action on skeletal muscle tissue, and review previous findings regarding the role of concentric and eccentric muscle actions on the development of strength and hypertrophy following dynamic constant external resistance (DCER) training.

Differences between Concentric and Eccentric Muscle Actions

Maximal strength

There are differences between concentric and eccentric muscle actions in terms of the maximal force that can be produced. The speed of movement has a large impact on maximal concentric force. As the velocity of concentric muscle actions increases, maximal force decreases (Behm & Sale, 1996; Bell & Wenger, 1992; Kellis & Baltzopoulos, 1998; Westing et al., 1991). Conversely, the maximal amount of force that a muscle can exert during eccentric muscle actions is largely unaffected by the velocity of movement (Kellis & Baltzopoulos, 1998; Oskowski et al., 1995; Westing et al., 1991). Maximal force during eccentric actions exceeds that of concentric actions at the same velocity of movement with observed differences in maximum force ranging from 20% at slower velocities to ~ 150% at faster velocities (Donne & Luckwill, 1996; Gulling et al., 1996; Kellis & Baltzopoulos, 1998; Oskowski et al., 1995; Westing et al., 1991).

Mechanical

The difference in maximal force between concentric and eccentric actions has been attributed to a mechanical rather than ATP-dependent cross-bridge detachment. During eccentric actions, the actin-myosin cross-bridges are pulled 'backward' due to the external force. The myosin heads are not allowed to rotate and the cross-bridges remain attached. Cross-bridge attachment continues to occur and the overall number of cross-bridges may be greater than during concentric actions. Alternatively, the myosin head may be mechanically pulled away from the actin binding site. This detachment produces tension in the muscle which is greater than that produced during the power stroke alone, as may occur during concentric actions (Stauber, 1989). The mechanical separation of cross-bridges can explain the characteristic shape of the eccentric force-velocity curve. The lower forces produced at slower eccentric velocities is thought to be related to additional time for cross-bridge cycling to occur, thereby reducing the net tension development.

Metabolic

It has long been known that eccentric actions are associated with a lower utilization of oxygen than concentric actions (Asmussen, 1953). Aura and Komi (1986) examined mechanical efficiency over a range of intensities using a leg press exercise. The efficiency of eccentric actions was much higher than during concentric actions, being 13-23% and 32-163% for concentric and eccentric work, respectively. The additional energy cost of adding eccentric actions to concentric actions during bilateral supine leg press was found to be only 14%, despite double the total number of muscle actions with the combined eccentric/concentric repetitions versus the concentric action only repetitions (Dudley et al, 1991a). This difference in efficiency and energy cost between concentric and eccentric actions might be explained in two ways. Firstly, the inherent capacity of skeletal muscle to develop greater tension during eccentric actions results in less cross-bridge cycling and thus ATP requirements are reduced. Secondly, the lower muscle activation found with eccentric muscle actions at various velocities and intensities implies that fewer fibres are active and therefore less oxygen is required.

Use of nuclear magnetic resonance spectroscopy provides a direct, non-invasive means to study muscle bioenergetics at rest and during exercise. Mechanochemical efficiency can be determined using the ATP production rate in the working muscle, rather than whole body energy costs, measured by oxygen uptake. This technique determined that for the workload and velocity examined, concentric activity was less efficient at 15% versus 35% for eccentric activity (Ryschon et al., 1997).

Neural

There are clear differences in neural activity with concentric and eccentric actions. A linear response of increased neural activity, measured by electromyography (EMG), with increased force is found with both actions, and can be attributed to recruitment and rate coding (Bigland-Ritchie, 1981; Sale, 1988). For a given force, lower levels of activation are required by the nervous system for eccentric actions (Bigland & Lippold, 1954; Nakazawa et al., 1992). This lower activity may be associated with comparatively fewer active motor units, indicating a higher tension per active motor unit (Bigland-Ritchie & Woods, 1976; Faulkner et al., 1993).

The effect of velocity of movement on muscle activity during maximal force generation also demonstrates clear differences in EMG between actions. The performance of eccentric actions at all velocities is consistently associated with lower muscle activation than concentric actions at the same velocity (Aura & Komi, 1986; Donne & Luckwill, 1996; Golden & Dudley, 1991; Gulling et al., 1996; Kellis & Baltzopoulos, 1998; Westing et al., 1991). While EMG has been shown to increase with increasing velocity of concentric isokinetic movement, velocity of eccentric actions during isokinetic movements does not appear to influence EMG activity (Enoka, 1996; Kellis & Baltzopoulos, 1998; Westing et al., 1991). This relatively constant level of EMG during eccentric actions at different velocities may be partially explained by mechanical differences in force development between eccentric and concentric actions.

Motor unit behaviour during slow repetitive dynamic anisometric concentric and eccentric actions also show differential responses between muscle actions (Nardone et al., 1989; Tax et al., 1989; Theeuwen et al., 1994). Increased motor units firing frequency (Tax et al., 1989) and lower recruitment thresholds for motor units (Tax et al., 1989; Theeuwen et al., 1994) during concentric actions can increase overall motor unit activity and explain the greater neural activity during concentric actions. Eccentric actions, even during very slow movements with a low submaximal load, may not precisely follow Henneman's size principle and fast-twitch motor units may be preferentially recruited (McHugh et al., 2002; Nardone et al., 1989).

Other biophysical factors can also explain the differences in neural activity between concentric and eccentric muscle actions. Specifically, the greater mechanical efficiency of eccentric actions during maximal and submaximal work (Aura & Komi, 1986), and lower metabolic cost of eccentric work (Bigland-Ritchie & Woods, 1974; Dudley et al., 1991b) can explain the lower muscle activation levels found with maximal and submaximal eccentric muscle actions at various velocities.

Since the classic investigation of Bigland and Lippold (1954), there has not been subsequent investigation regarding muscle activation and velocity with non-maximal effort. Recently, the EMG – velocity relationship with a submaximal allokinetic (dynamic) load during non-fatiguing conditions has been investigated (Gillies et al., 2000, Appendix A). Concentric and eccentric actions were examined over a range of velocities during plantar flexion with a load of approximately 25% of isometric maximum. Eccentric neural activity was found to be similar to the response reported with maximal isokinetic eccentric loads; the average EMG (aEMG) of eccentric actions was lower than concentric actions at the same velocity. As well, velocity of eccentric movement demonstrated no impact on aEMG. In contrast, during concentric movements, there was a positive relationship between velocity and EMG of the agonist muscles. However, when total muscle activation was considered, total EMG was greater during the slower than the faster movements for both concentric and eccentric muscle actions, despite the lower aEMG of the slower movements. The results of this investigation are interesting as they may indicate that not only muscle action, but also the velocity at which a muscle action is performed, may impact on total muscle activation and thus potential neuromuscular adaptations following dynamic resistance training.

Muscle Damage/Injury following Eccentric and Concentric Muscle Actions

Ultrastructural indication of muscle damage

Ultrastructural examination of muscle fibres following resistance type exercise, especially involving protocols with maximal eccentric muscle actions, clearly reveal that damage to the fibre structure does occur. This is evidenced by disruption of the orderly arrangement of the cellular components, especially along the Z line region, resulting in widening of the Z-lines, Z-line smearing, sarcomere misalignment and disruption of the myofilament structure within the sarcomere (Fiatarone Singh et al., 1999; Hortobagyi et al., 1998; Newham et al., 1983; Roth et al., 1999, Roth et al., 2000; Waterman-Storer, 1991). Muscle disruption and damage is not limited to maximal eccentric actions but also occurs subsequent to submaximal eccentric actions (Gibala et al., 1995), as employed during DCER training, although muscle damage is attenuated with prior resistance training experience (Gibala et al., 2000).

Prior bout of eccentric exercise and muscle damage with continued resistance training

It has been demonstrated that an acute bout of maximal eccentric exercise producing muscle damage may provide a prophylactic effect against muscle damage with subsequent eccentric exercise (Clarkson et al., 1992; Clarkson & Tremblay, 1988; Hortogabyi et al., 1996; Nosaka and Clarkson, 1995; Newham et al., 1987). It must be considered that the eccentric protocols employed in those investigations were quite extreme and not the same as would be encountered with continued resistance training sessions. However, it has been shown that muscle damage continues to occur with on-going training, or is greater post-training than pre-training using both direct (Fiatarone Singh et al., 1999; Roth et al., 1999, 2000; Staron et al., 1992) and indirect (Frontera et al., 1988; Nissen et al., 1996) determination of muscle damage. As well, Fiatarone Singh et al. (1999) found that despite a large increase in ultrastructural damage following resistance training, significant increases in strength and fibre hypertrophy also occurred. This suggests that muscle damage/degeneration is a mechanism for skeletal muscle hypertrophy.

Markers of muscle damage

The presence of elevated levels of intramuscular proteins in the blood or urine is taken as indication that damage has occurred to the muscle cell structures along with a loss of some cell membrane integrity, which allows for the efflux of these proteins. Some of the intramuscular proteins that have been measured in blood include myosin heavy chain (MHC) fragments (Prou et al., 1999, Sorichter et al., 1997a), troponin I and myoglobin (Hayward et al., 1997; Sorichter et al., 1997b). The most widely used blood marker of muscle damage, however, is creatine kinase (Ebbeling & Clarkson, 1989).

Three-methylhistidine (3-MH) is a urinary indicator of skeletal muscle degradation (Frontera et al., 1988; Hickson et al., 1986; Nissan et al., 1996; Paul et al., 1989; Viru & Seli; 1992). 3-MH, a component of actin and myosin, is a marker of myofibrillar protein degradation as it is not reutilized by muscle tissue or catabolized by body tissues and must be excreted in the urine (Young and Munro, 1978). Dietary intake of animal and fish flesh and stock influences 3-MH urine content and a 48 hour meat free diet is necessary to clear exogenous sources of 3-MH (Huszar et al., 1983).

Estrogen and Muscle Cell Membrane Integrity

Recent investigations have demonstrated that estrogen levels may exert a protective effect on skeletal muscle cell membrane integrity resulting in less muscle damage, as evidenced by lower levels of markers of muscle damage (e.g. creatine kinase) and a lesser degree of delayed onset muscle soreness subsequent to resistance training (Hayward et al., 1997), or reduce histological indices of damage (Komulainen et al., 1999). Roth and co-investigators have recently demonstrated that following identical resistance training protocols in humans, muscle damage in young adult women did not increase following resistance training while muscle damage was increased in older women and young and older men (Roth et al., 1999; Roth et al., 2000). These results suggest that estrogen may play a role regarding muscle damage during resistance training. Proposed mechanisms for this response is that estrogen may exhibit antioxidant properties or may bind to Vitamin E receptors on the sarcolemma. Vitamin E is a known anti-oxidant that stabilizes cell membranes and affords protection from damage (Tiidus, 2000).

The influence of higher levels of estrogen, as are observed during the luteal phase of normal menstrual cycles, and the effect of resistance exercise on muscle damage, in comparison to the follicular phase, has not been investigated. Menstrual status of females is rarely reported in the literature. It remains to be explored if the response to resistance training of females of normal menstrual status differs with their follicular and luteal phases, or if females of different menstrual status, such as amenorrhea, oligomenorrhea or pharmacological contraceptive usage, display a differential response to resistance training. However, studies that have specifically examined gender response following the same resistance training program have clearly demonstrated that the response of females over the duration of these investigations, in terms of increased strength and muscle hypertrophy, does not differ from that of males (Colliander & Tesch, 1990; Cureton et. al., 1988; O'Hagan et al., 1995; Staron et al., 1994; Weiss et al., 1988).

Exosarcomeric Muscle Proteins

Pathological changes in muscle morphology following damage certainly suggests that the structural framework of the cell has been disrupted. Many of the disruptions are associated with the Z line and indeed, alterations in the associated cytoskeletal proteins associated with the Z line occur following muscle damage induced through exercise (Fridén et al., 1984; Komulainen et al., 1999) electrical stimulation (Komulainen et al., 2000, Lieber et al., 1996) or chemical-induced fibre necrosis (Bornemann & Schmalbruch, 1992).

The intermediate filaments (IF) are arranged longitudinally and transversely around the myofibrils (Tokuyasu et al., 1983; Waterman-Storer, 1991) and are responsible for registry of the sarcomeres (Franzini-Armstrong & Fischman, 1994) as well as transmission of the force generated by the contractile proteins (Patel & Lieber, 1997). The primary protein of the IF is desmin (Tokuyasu et al., 1983). Desmin is also one of the major proteins of the Z line (Patel & Lieber, 1987). Dystrophin is another endosarcomeric protein that has received attention. In part this is due to its association with primary myopathies. Dystrophin appears to play a role in membrane stability, as dystrophic muscle demonstrates progressive degeneration and displays increased susceptibility to contraction-induced injury (Petrof, et al., 1993; Lynch et al., 2000). Investigations into alterations in non-contractile protein expression in response to exercise are limited, especially with a human training model, with the exception of Fridén and co-workers (1984). In part, this can be attributed to the fairly recent identification of some of these proteins.

There is some suggestion that fast twitch fibres are more susceptible to exercise-induced damage (Fridén et al., 1983; Lieber and Fridén, 1993; Macpherson et al., 1996).

The Z line width is narrower in fast twitch than in slow twitch fibres (Fridén et al., 1988; Schachat et al., 1988; Takekura & Yoshioka, 1990), which may explain increased susceptibility to damage of fast twitch fibres. The proteins of the Z line are dynamic and do respond to damaging protocols, especially eccentric action. Immumohistochemical evidence suggests that Z line and/or IF reorganization precedes that of shifts in contractile proteins (Komulainen et al., 2000; Lieber et al., 1996; Schachat et al., 1988). Baldi and Reiser (1995) demonstrated increased desmin and vimentin expression following chronic low-frequency electrical stimulation of rabbit muscle. They suggested that increased IF proteins in conjunction with alterations in the Z line indicate that 'fortification of the force-bearing ultrastructure' can occur.

Theories Regarding Mechanisms of Muscle Hypertrophy with Resistance Training

The exact mechanisms by which skeletal muscle enlarges, or hypertrophies, are not clearly understood. There are several theories regarding proposed mechanisms, namely a tension or mechanical stimulus mechanism, metabolic mechanisms, and muscle degeneration/regeneration. The tension/mechanical theory suggests that a certain level of repeated muscular tension is required for muscle hypertrophy. Indeed, tension is a critical factor regarding hypertrophy since muscle increases in size in response to tension, or the applied load (Goldberg, et al. 1975). Muscle actions of low force or tension development (less than 30% of maximal strength) are not necessarily conducive to the development of maximal strength (Anderson & Kearney, 1982; Dons et al., 1979; Häkkinen et al., 1985) and often provide an insufficient stimulus for muscle hypertrophy (Dons et al.; Sale & MacDougall, 1981). Muscle action may also be an important consideration regarding tension. While muscle hypertrophy can occur with the use of maximal concentric tension (as developed with an isokinetic mode) (Narici et al., 1989), the same is not always found with an isokinetic eccentric mode (Farthing & Chilibeck, 2003).

It has also been theorized that metabolic events that occur during resistance training provide the stimulus for hypertrophy. With a reduced short-term energy status in the working muscle, there are alterations in muscle pH and the accumulation of metabolic by-products. Resistance exercise protocols with a greater metabolic demand, or resulting in a greater accumulation of phosphate metabolites and a lower pH, have been linked to greater hypertrophy (Carey Smith & Rutherford, 1995: Schott, 1995). As well, the increased metabolic demands of the muscle results in systemic support with increased hormonal action and protein synthesis, resulting in a net result of increases myofibrillar proteins. In addition to enhanced protein synthesis following resistance exercise (Hernandez et al., 2000), reduced muscle proteolysis (Villani, 1987) may also be important for net protein accumulation and subsequent hypertrophy.

A final theory for muscle hypertrophy suggests that muscle degeneration followed by regeneration ('break down – build up') is the stimulus for hypertrophy. Regeneration, or the repair of damaged fibres may be accompanied by an 'overshoot' of protein synthesis and increased muscle fibre size. Satellite cell activity is considered to important to the repair process in skeletal tissue (for review, Hawke & Garry, 2001). Evidence to support degeneration/regeneration as a mechanism for hypertrophy comes from findings of muscle damage following resistance training protocols that have also induced hypertrophy (Fiatarone Singh et al., 1999; Kadi & Thornell, 1999), as well as increased satellite cell content (Kadi & Thornell, 2000).

It is highly likely that the different mechanisms of hypertrophy related to the different theories are inter-related. The signaling pathways involved with skeletal muscle cell hypertrophy are varied. Altered intracellular calcium balance, as can occur with eccentric actions and with muscle damage, can trigger the signaling pathways of calcineurin and its activation of transcription factors (nuclear factor of activated T cells) leading to hyperprophy (Dunn et al., 1999). There are a number of growth factors associated with satellite cell regeneration of muscle fibres. These include insulin-like growth factors, hepatocyte growth factor, fibroblast growth factors, transforming growth factors and interleukin-6 cytokines, each with their own signaling pathways (Adams & Haddad, 1996; Hawke & Garry, 2001). While the specific role of each of these growth factors regarding hypertrophy in response to resistance training and with different muscle actions remains to be elucidated, it is clear that they are involved with satellite cell regulation.

Cortisol Action on Skeletal Muscle Tissue

While the primary role of cortisol is a permissive action on gluconeogenesis in the liver, the action of cortisol on skeletal muscle tissue is considered to be catabolic in nature due to stimulation of muscle proteolysis (Hadley, 1996; Viru et al., 1994). Thus elevated levels of cortisol may adversely affect skeletal muscle hypertrophy and reflect overall metabolic stress. While elevated cortisol levels in women following a resistance training program with TUT not controlled or specified has not been reported (Bell et al., 1997; Bell et al., 2000; Staron et al., 1994), there is interest in examining cortisol levels as elevated cortisol may be a marker of training stress or overtraining (Urhausen et al., 1995) and may lead to subsequent decreased performance.

Strength and Hypertrophy Responses with Concentric and Eccentric Muscle Actions

It is well established that training with DCER, or 'free weights', results in strength gains as well as muscle hypertrophy. With DCER, the eccentric resistance is often determined by the concentric resistance (subjects raise and lower the same mass). The eccentric load may be increased through unilateral eccentric actions combined with bilateral concentric actions, assistance in completing the concentric phase, or additional load applied for the eccentric action. Despite the popularity of DCER as a training mode, very few studies have evaluated the role of concentric and eccentric muscle actions in DCER training. Concentric and eccentric actions have been evaluated with the load for both actions based on the submaximal concentric resistance (Adams et al., 1993; Ben-Sira et al. 1995; Dudley et al., 1991a; Hather et al., 1991; Johnson, 1972) and with the eccentric load greater than the concentric load (Ben-Sira et al., 1995; Brandenburg & Docherty, 2002; Carey Smith & Rutherford, 1995; Häkkinen & Komi, 1981; Johnson et al., 1976; Seliger et al. 1968). Three of these investigations were based on the same subjects and reported the effects of the training program on strength response (Dudley et al., 1991a), muscle fibre type changes (Hather et al., 1991) and myosin heavy chain alterations (Adams et al., 1993). All of these studies used similar sets and repetitions, which minimizes the impact of comparing largely varying training regimens. With the exception of Ben-Sira et al. (1995), all subjects were male.

Strength following DCER training with equal loads for concentric and eccentric actions

There are few reports on the role concentric and eccentric muscle actions play in strength development. Johnson (1972) examined the effects of training with either concentric or eccentric only actions in both bench press and leg extension, with the training resistance set at 80% of concentric maximal strength. Maximal strength was assessed by 1 repetition maximum (1 RM) bench press and leg extension, although it was not specified whether this included both concentric and eccentric actions. Both groups had similar and significant increases in 1 RM for both exercises (41-54%).

The investigations of Dudley et al. (1991b) and Ben-Sira et al. (1995) differed from the study of Johnson (1972) in that combined eccentric and concentric actions were also examined. Dudley and co-investigators (1991b) examined the effects of coupled eccentric and concentric actions versus concentric only actions. Concentric only actions were performed with an equal number of sets and repetitions as the coupled actions, or were matched to the total number of actions performed by the coupled action group (i.e. twice the number of sets). Both exercises performed during training were tested with a combined eccentric/concentric test and a concentric only test. In general, the results indicated that the combined action group demonstrated the greatest increases in strength of the different tests, and with concentric only actions, more repetitions enhanced strength.

Ben-Sira and co-workers (1995) also compared strength increases following training with concentric and/or eccentric muscle actions and reported that there was no difference in terms of concentric strength following training. Training intensity was set at 65% of concentric 1 RM. Groups trained with an equal number of repetitions of coupled concentric/eccentric actions, eccentric actions, or concentric actions, and thus the coupled action group completed twice the number of actions as the other groups. All training groups experienced significant increase in strength, with no significant differences occurring between training groups. As subjects trained only twice weekly for 8 weeks, a greater frequency of training, a greater training intensity, or a longer training period might have revealed significant differences between the groups.

In summary, the inclusion of eccentric actions with the load based on a percentage of maximal concentric strength appears to maximize strength increases, especially when strength is tested with combined actions. While eccentric actions alone can enhance concentric strength, it is not clear if training with combined eccentric/concentric actions augments concentric strength more than training with concentric actions only. These conclusions are tenuous in that they are based on a limited number of studies. Further examination of the role of concentric and eccentric muscle actions using the same resistance, and the resultant effects on dynamic strength, assessed by concentric, eccentric, or combined eccentric/concentric modes is clearly required.

Hypertrophy and alterations in MHC isoforms following DCER training with equal concentric and eccentric loads

While investigation into the effects of concentric and eccentric actions, in isolation or combined, on muscular strength development is limited, there is even less available information on hypertrophy and alterations in MHC expression. Hather and co-workers (1991) reported on the effects of training with coupled concentric and eccentric actions or concentric only actions on alterations in vastus lateralis muscle fibre cross-sectional area (CSA). Both the combined action group and the group that performed an equal number of concentric actions only displayed significant increases in type-II fibre CSA while the combined action group only displayed had significant hypertrophy of type-I fibres. The concentric group that trained with one-half the number of concentric only actions, and the control group demonstrated no significant hypertrophy. Based on this one study, it appears that hypertrophy is optimized with the inclusion of eccentric actions and that a certain volume of training must be performed to observe hypertrophy. Ben-Sira et al. (1995) assessed hypertrophy by an indirect method of girth measurement and found no significant change in thigh girth for any of the groups. However, it is difficult to draw any conclusions using this method.

Adams et al. (1993) examined alterations in MHC isoforms following concentric and coupled concentric/eccentric actions. The results for both types of training were comparable and thus the results were combined. Transformation within the fast MHC isoforms were noted, characterized by a decreased proportion of MHCIId(x) and a concomitant increase in MHCIIa. No changes in the proportions of slow MHCI were found, suggesting that resistance training did not result in the transformation between fast and slow MHC isoforms. These changes in MHC isoforms were the same as the alterations in fibre type distribution. It must also be noted that the training duration of this study was 19 weeks, which may have been of sufficient duration to observe agreement between histochemically determined fibre types and alterations in MHC proportions.

Strength following DCER training with a greater load for eccentric actions

The effects on strength of training with DCER exercise with the eccentric load greater than the concentric load has also been investigated. Johnson et al. (1976) trained subjects with either concentric only actions with the load set at 80% of concentric 1 RM, or with eccentric actions of 120% of concentric 1RM. Both modes of training were equally effective at significantly increasing isometric and DCER strength. Ben-Sira and co-workers (1995) compared strength increases with bilateral concentric or unilateral eccentric muscle actions and also reported that the greater eccentric load did not enhance concentric strength to a greater degree than concentric actions of one half the load. Similarly, Seliger et al. (1968) found that training eccentrically with 145-150% of concentric 1 RM did not augment concentric strength to a greater degree than using concentric loads of 90-95% of 1 RM. It has also been reported that training with concentric 10 RM loads results in greater increases in isometric strength than eccentric training with a 35% greater load (Carey Smith & Rutherford, 1995). These results suggest that concentric strength is not enhanced by training with eccentric loads greater than maximal concentric strength. However, Häkkinen and Komi (1981) reported that the inclusion of some eccentric training with loads of 100-130% of concentric 1 RM resulted in significantly greater increases in strength than concentric only training in some of the strength tests. Seven different strength tests were performed which included training specific exercises as well as exercises assessed with isometric and isokinetic test modes. This difference may be related to the inclusion of both concentric and eccentric actions during training and/or the different test modes.

Hypertrophy following DCER training with a greater load for eccentric actions

There is a lack of investigations regarding the effects of training using concentric actions based on a submaximal concentric load and eccentric actions with a greater load (i.e. augmented eccentric) on hypertrophy. Carey Smith and Rutherford (1995) measured whole muscle hypertrophy using computer tomography and found that there was no difference in hypertrophic responses following both concentric and augmented eccentric training. It has also been reported (Brandenburg & Docherty, 2002) that augmented eccentric training load may in fact have a detrimental effect on muscle growth.

Conclusions regarding concentric and eccentric muscle actions during DCER training

In dynamic resistance training with the resistance based on a sub-maximal concentric resistance, the inclusion of eccentric muscle actions appears to be important for optimizing strength performance and hypertrophy. However, the inclusion of eccentric loads greater than concentric 1 RM does not necessarily lead to an enhanced response. Strength development needs to be more fully examined as it has largely been examined in a concentric only mode. As there is a lack of investigations that have examined the role of concentric and eccentric muscle actions with regards to muscle hypertrophy and MHC expression, further research is needed to clearly define the relative contributions of these muscle actions on fibre hypertrophy and patterns of MHC expression as phenotypic parameters that underlie some aspects of strength development in response to dynamic resistance training.

CHAPTER II - STRENGTH, MUSCLE MORPHOLOGY, MYOSIN ISOFORMS AND CORTISOL RESPONSES FOLLOWING RESISTANCE TRAINING WITH MANIPULATION OF CONCENTRIC AND ECCENTRIC MUSCLE ACTIONS IN RECREATIONAL RESISTANCE-TRAINED WOMEN.

Introduction

Resistance training is a popular form of exercise performed for benefits related to health, rehabilitation, occupational and personal fitness, and athletic performance. A major outcome of resistance training is increased muscular force, which may be accompanied by an increase in muscle mass or cross-sectional area (CSA) (hypertrophy). This increase in muscle size is often a desired outcome of resistance training as muscle CSA is directly related to potential increased force production.

Muscle hypertrophy following resistance training can be assessed through whole muscle imaging techniques (i.e. computer tomography, magnetic resonance imaging, or ultrasound) or analysis of individual fibres using a muscle biopsy. In recent years, there has been much interest in the examination of changes that occur with specific muscle proteins, especially myosin heavy chain (MHC) isoforms. Specific MHC isoforms, and alterations in these isoforms are of interest as they are reflective of the functional demands imposed on skeletal muscle tissue (for review see Pette & Staron, 2000).

Concentric and eccentric muscle actions may be employed to various degrees with different resistance training regimens using a human training model. The underlying mechanisms and rôles in which different muscle actions play in strength and hypertrophy development are still not completely understood. It is clear that there are differences between concentric and eccentric in terms of maximal force, muscle damage, metabolic energy cost, and neural activation. It is less clear with respect to the overall response with respect to strength development and muscle hypertrophy, in consideration of the interaction of these factors, and with consideration of the potential resultant effects of manipulating the time under tension (TUT), or the duration of muscle action, for concentric and eccentric muscle actions.

Purpose of the Study

The purpose of this study was to investigate the effects of resistance training with different times for the concentric and eccentric muscle actions, with the total TUT equated for both actions, on maximal strength development, alterations in muscle morphology, muscle protein expression and cortisol levels. A secondary purpose was to examine the response of acute resistance training variables over the duration of the training period.

Statement of problem and significance of study

The role of the time that muscle is under tension on strength and hypertrophy development has remained relatively unexplored with respect to DCER training modalities. Isometric and isokinetic training and testing investigations clearly specify the TUT, the range of movement or the angle of application of force, and often specify the effects of various test velocities on maximal force development. In general the effects of training velocity using isokinetic movements, including both concentric and eccentric actions, are such that the greatest increases in strength are observed at or near the training velocity (Bell & Wenger, 1992).

Recently, it has become more common for training studies using a DCER modality to specify TUT for concentric and eccentric muscle actions (i.e. the duration in s to complete the full range of movement of the muscle action) for concentric and eccentric muscle actions (Baker, 1995: Ben-Sira et al., 1995; Brandenburg & Docherty, 2002; Dudley et al., 1991b; Frontera et al., 1998; Gillies & Docherty, 1999; Morrissey et al., 1998; Palmieri, 1989; Schott et al., 1995; Weir et al., 1995;Young & Bilby, 1993), and thus the effects that movement velocity during DCER resistance-type exercise may have on subsequent adaptations can be controlled or allow comparisons between protocols.

There are a few studies that have manipulated concentric TUT as an individual training variable (Gillies & Docherty, 1999; Morrissey et al., 1998; Palmieri, 1989; Young & Bilby, 1993). In 3 of these studies, all muscle actions were completed in 2 s or less (Morrissey et al.; Palmieri; Young & Bilby) while Gillies and Docherty used 2 s and a longer concentric action time of 8 s, and also equated training volume between groups. All of these investigations reported equivocal effects on combined strength. However, Gillies and Docherty also reported significantly greater muscle hypertrophy with the slower concentric protocol.

The influence of differential TUT for eccentric actions on strength and hypertrophy remains unexplored with a DCER training mode. In general, the role of TUT, for concentric and eccentric muscle action, especially with very slow movements, and the resultant neuromuscular adaptations, has clearly not been adequately investigated.

There is evidence suggesting that TUT may be an important consideration regarding strength and hypertrophy development. It is apparent to resistance training practitioners that a decreased average velocity of movement during training necessitates a reduction in training load. However, this may not be true with continued training. A recent investigation demonstrated that for slow concentric actions, this effect is transitory, and that greater increases in training load for each set occurred with the training group that employed very slow concentric actions as opposed to more "typical" durations of concentric muscle actions (Gillies & Docherty, 1999). The results of that investigation also suggested that muscle hypertrophy may be maximized with the slow movements, although this remains to be substantiated. Mikesky et al. (1989) used an animal weight lifting model and found that the two variables that correlated the highest with whole muscle hypertrophy were the absolute load and a longer length of time to complete the movement. Together these findings suggest that not only absolute tension, but also the time that the muscles are under tension during the muscle action are important to maximize strength and hypertrophy.

Hypotheses

It was hypothesized that increased strength would occur with both training regimens. As well, it was hypothesized that manipulation of the time to complete the concentric and eccentric actions would be associated with a differential response in the muscle. Increased strength following training with a long concentric action would coincide with hypertrophy of both type-I and -II muscle fibres, with greater hypertrophy of the IIa fibres, whereas increased strength following training with a long eccentric action would be accompanied by hypertrophy of type-IIa fibres only. It was also hypothesized that there would be a decrease in the proportion of MHCIId(x) following both training regimens along with a corresponding increase in MHCIIa.

Limitations

1. Expense and time considerations limited the number of muscle biopsies obtained and thus limited the subsequent analyses involving skeletal muscle tissue.
2. Individual variability in the training response may have occurred due to between subject differences in resistance training experience, fibre type proportions and muscle ultrastructure which could have influenced the training response.
3. Other physical activities, including daily functional demands, were not strictly controlled and could have influenced the training response.
4. Caloric intake and macronutrient proportions were monitored for 3-4 days only every two weeks.
5. The relatively small sample size and female only subjects may not allow for generalizations to be made to the global population.
6. The subjects who participated were derived from the University of Alberta and surrounding community. Therefore, true random selection of the population was not achieved and generalizations to other populations may not be appropriate.

Delimitations

1. All subjects were healthy adult females aged 19 to 38 years.
2. The individual resistance training experience varied between subjects.
3. Muscle tissue sampling was from the vastus lateralis muscle only.
4. Muscle biopsies were obtained pre- and post-training only.
5. Supine leg press strength was the primary strength measure.
6. The training duration was nine weeks only.
7. Twenty-four hour urine was collected at the beginning and end of the training period.

Methods

Subjects

Twenty-nine women with current resistance training experience, defined as adhering to a program of multiple sets of various upper and lower body exercises performed with 8-12 repetitions to failure, 2-3 times per week for a minimum of 8 weeks prior to recruitment to the study, volunteered to participate in this study. Following recruitment to the study, all subjects continued with their usual resistance training regimen albeit at a frequency reduced to 1-2 times per week for 4-6 weeks before commencing training. This was done to coordinate the start of the training program with individual menstrual cycles. Of the 29 initial subjects, 28 completed all training and testing requirements. These 28 subjects had a mean (SD) age of 24.3 (5.8) yr, height of 164.6 (7.3) cm, body mass of 66.7 (9.2) kg, BMI of 24.7 (3.7) kg • m^{-2}) and body fat of 17.6 (5.0) % [prediction equation using sum of 6 skinfolds (tricep, subscapular, iliac crest, abdomen, front thigh, rear thigh (Yuhasz, 1966)]. Thigh girth was determined as the circumference of the thigh at same location at which the front and rear thigh skinfolds were obtained. Written informed consent was obtained from all subjects and the research was cleared by the Faculty of Physical Education and Recreation Research Ethics Committee at the University of Alberta.

Experimental Design

This study used a repeated measures design in which subjects were randomly assigned to one of two resistance training regimens. The total duration of the experimental period was 11 weeks. Scheduling of all assessments is outlined in Table 1. Subjects refrained from resistance training, including strength testing sessions, and other physical training for 3-4 days prior to obtainment of both pre- and post-training muscle biopsies. As well, there were 4 days scheduled between resistance training sessions and maximal strength testing. An exercise familiarization session occurred prior to the first training session. After initial maximum strength testing (1 RM), subjects were stratified according to menstrual status of no pharmacological contraceptive use or pharmacological contraceptive use (i.e. pills, patch or injection - all of these subjects used oral contraceptives). Within each group, subjects were ranked in order by initial 1 RM combined eccentric-concentric leg press strength and each consecutive pair of subjects was randomly assigned to one of the two training protocols. Subjects using no pharmacological contraceptives commenced training between days 4-6 of their menstrual cycle (i.e. early follicular phase) while those using oral contraceptives began training during days 22-24 of their cycle, with day 1 commensurate with pill number 1. Thus the onset of training for all subjects was coordinated with low estrogen levels.

Resistance Training Protocols

Both groups were matched for the total time under tension (TUT) defined as the time to complete the concentric muscle action plus the time to complete the ECC action, along with a 1 s pause between actions. The Long Concentric (LC) group performed the concentric action in 6 s and the eccentric action in 2 s, while the Long Eccentric (LE) group performed the concentric action in 2 s and the eccentric action in 6 s. This is based on average velocities of approximately 0.2 and 0.8 rad $\cdot$ s $^{-1}$. These velocities were chosen in order to attempt to match the total neural activation during both muscle actions, and thus minimize or equalize the effects of muscle activation related to the velocity of the muscle actions (see Appendix A).

Six exercises were performed during each training session. During each session, four lower body exercises of bilateral incline leg press, parallel squat, bilateral leg extension, and leg flexion were performed. In addition, bench press was completed during each session while the final exercise of bilateral bicep curl, lat pull down or seated row was alternated between subsequent sessions. All lower body exercises were completed first in the order listed above, followed by the 2 upper body exercises.

Subjects performed two sets of each exercise with a resistance that elicited technical failure within 6-8 repetitions (6 – 8 RM) with 2 ½ minutes of rest between sets. The load was progressively increased approximately 2.5 - 5 % when subjects were able to complete 8 or more repetitions during the second set of an exercise. Each training and testing session was preceded by a 5 min warm-up on a cycle ergometer and 5 min of stretching. A metronome set to 60 beats per minute was used to aid the timing of the muscle actions. Subjects recorded the training load and repetitions for each set of all exercises in training logs. All training sessions were monitored to ensure adherence to the training protocols and to help subjects determine when changes in load were necessary to keep the repetitions within the desired repetitions to failure.

Training sessions occurred 3 times a week over a 9 week period, with the exception of 2 training sessions only during the first week and 1 training session only during the fifth week of the program, for a total of 24 training sessions. Sessions during the fifth week consisted of 1 training session, a "rest" day and a 1 RM testing session. This gave a total of 12 training sessions both before and after the mid-training strength test.

Strength Assessment

Maximal strength (1 RM) was assessed prior to training, mid-training and post-training, using modified procedures of Dalton and Wallace (1996). Modifications were as follows: the increase in load between subsequent 1 RM attempts for bench press was approximately one-quarter the load for leg press; the previous 1 RM value was used rather than an estimated value after the initial strength test; the initial estimated leg press 1 RM was 3 times body mass; and the initial estimated bench press was 75 % of body mass. 1 RM concentric, eccentric and combined eccentric/concentric strength were tested using incline bilateral leg press and bench press exercises. During the leg press tests, the range of movement (ROM) was 1.57 rad extension (with full extension = 0 rad) while the ROM during bench press was from full extension of the arms to the chest. The concentric only test started with a knee joint angle of 1.57 rad for leg press, or with the load at the chest for bench press, and went to full extension. The load was fully supported at the start position by fixed supports for leg press, or by assistants for bench press. During concentric 1 RM bench press, following a verbal command from the subject, the load was released, the subject momentarily supported the load and then the subject ‘lifted’ the load. Eccentric 1 RM was assessed by gentle release of the load by assistants, verbal indication by the subject that she had full support of the load at full extension, and then lowering of the load under control throughout the ROM over a 3 s duration. The combined eccentric/concentric test involved lowering the load and then raising it to full extension. Failure during the concentric and combined eccentric/concentric 1 RM attempts was determined by inability of the subject to achieve full extension while failure during the eccentric 1 RM test was determined when the subject was unable to control the descent throughout the full ROM.

Verbal encouragement was given during all 1 RM attempts and subjects were instructed to "explode up as hard and fast as possible" during the concentric actions of the concentric and combined action tests and to "maintain control and lower the load with an even velocity" throughout the ROM during the eccentric strength tests. Five minutes of rest was taken between subsequent 1 RM attempts. Test order for the concentric, eccentric and combined tests were randomized for the pre-training session and was maintained for subsequent tests. Leg press strength was assessed prior to bench press strength. The intraclass correlation coefficient for 1 RM strength of 5 subjects tested 3-5 days apart was; R=.986, R=.994, and R=.965, for combined, concentric and eccentric 1 RM, respectively.

Relative Training Load (% 1RM)

The relative training load was determined as the mean training load during the second training session of week 1 and the last training session load during weeks 5 and 9, and expressed relative to the 1RM combined strength of each respective 1 RM test.

Weekly Repetitions per Set

The weekly mean number of repetitions per set was determined as the mean number of repetitions completed during all sets of an exercise during each week.

Weekly Training Load

Training load for each week was determined as the mean load utilized during all sets of each exercise.

Training Volume

Training volume for each week was calculated by the sum of load x repetitions for each set.

Muscle Sampling

Muscle samples were obtained pre- and post-training using the needle biopsy technique adapted for suction (Evans et al., 1982). Tissue samples were taken from a site approximately one-third of the length from the proximal lateral edge of the patella to the anterior superior iliac spine of the lateral aspect of the left vastus lateralis muscle. The samples were oriented and mounted on cork in embedding medium (OCT, Tissue Tek, Miles Laboratories, Naperville, IL), quickly frozen in melting isopentane (-159 °C) precooled in liquid nitrogen and stored at –80 °C until analysis. The post-training biopsy was obtained at a new site 0.75 mm distal to the original incision. Muscle biopsies of 2 subjects (1 from each training group) contained insufficient muscle protein content for analysis and thus all analyses regarding muscle fibre CSA, fibre type proportions and MHC isoform content is based on 26 subjects only (LC, n=14; LE, n=12).

Immunohistochemistry for Myosin Fibre Typing

Serial 8-micrometer thick transverse sections were cut at –20 ° C in a cryostat (Tissue Tek, Miles Laboratories, Elkhart, IN), mounted on poly-L-lysine coated slides (Cedarlane Laboratories, Hornby, ON, Canada) for analysis of fibre type identification. Pre- and post-training samples of each subject were mounted and assayed on the same slide to avoid interassay variation. Immunohistochemical methods were used to classify fibre types based on myosin heavy chain (MHC) isoform content. The following monoclonal antibodies (mAb) directed against adult MHC isoforms were used: MHCI (clone BA-D5), MHCIIa (clone SC-71), MHCIIb (clone BF-F3). As well, mAb directed against MHCembryonic (clone BF-45) was used to detect of muscle fibre regeneration.

Fibre types were identified using modified procedures outlined by Putman et al. (2000). Briefly, the slides with the tissue sections were air-dried, washed once in PBS-Tween (0.1% vol/vol, pH 7.4), twice in PBS and incubated for 15 min in 3% H_2O_2 in methanol. Sections were subsequently washed as before and incubated overnight in a humid chamber at 4°C with a blocking solution [(BS) 2.4% bovine serum albumin, 6% horse serum, 0.1% Tween-20 in PBS] plus 4 drops of Avidin D complex (Blocking Kit, Vector Laboratories, Burlingame, CA) per ml BS. Excess BS-Avidin D was removed and the primary mAb, along with 4 drops of biotin solution (Blocking Kit, Vector Laboratories) per ml of primary Ab was overlaid and incubated for 1 hr at room temperature. Primary antibodies were diluted in BS as follows: MHCI (clone BA-D5), 1:200; MHCIIa (clone SC-71), 1:500; MHCIIb (clone BF-F3), 1:100; MHCembryonic (clone BF-45), 1:500. Sections were washed as before and incubated for 1 hr at room temperature with biotinylated horse anti-mouse IgG [diluted 1:400 in BS (IgM for BF-F3) secondary antibodies, Vector Laboratories). Sections were then washed, incubated with an avidin-biotin-horseradish peroxidase complex (Vectastain Elite ABC, Vector Laboratories) for 45 min at room temperature, washed again, and reacted for 6 min with a peroxidase diaminobenzidine [DAB/ $NiCl_2$ solution (DAB/Ni Substrate Kit, Vector Laboratories)]. The reaction was stopped by washing several times with distilled water. Sections were subsequently dehydrated in ethanol, cleared and mounted with Entellan (Merck, Darmstadt, Germany). Negative controls were completed in parallel by omitting the primary mAb with incubation in BS only. For mAb BF-F3 and IgM (biotinylated goat anti-mouse, 1:400 in BS) goat serum was substituted for horse serum in the BS.

Morphometric Analysis

Serial sections immunohistochemically stained for various MHC isoforms were visualized with a computer inter-faced light microscope (Leitz Diaplan, Ernst Leitz Wetzlar GmbH, Wetzlar, Germany) for analysis. Pure fibre types were identified by staining positive for each respective mAb, and negative for all others. Mixed type-I/IIA fibres were identified as staining positive for both MHCI and MHCIIa. As no mAb for human MHCIId(x) was available, type-IID(X) fibres were identified as staining negative on all slides. Cross-sectional area measurements were made using a semi-automated image-analysis software program (Image-Pro Plus, Media Cybernetics, Silver Springs, MD) with the same magnification for all analyses. All fibres within a given section were used to determine fibre type proportions (for all subjects combined; mean, SD, minimum-maximum: 695, 374, 101-1705 fibres/sample) while CSA measurements were made on 1-2 images of the section (~ 60-100 fibres per image) or on all fibres of one type if there were less than 50 fibres of a type. For all subjects combined, CSA was measured on (mean, SD, minimum-maximum number of fibres) 65, 21, 12-123 type-I fibres; 58, 24, 20-151 type-IIA fibres; 2, 5, 0-24 mixed I/IIa fibres; and 0, 1, 0-8 type-IID(X) fibres.

Muscle Protein Preparation

Muscle tissue of 5–10 mg was pulverized, weighed, and 6 times as much cold extraction buffer was added (100 mM $Na_4P_2O_7 \cdot 10H_2O$, 5 mM EGTA, 5 mM $MgCl_2 \cdot 6H_2O$ and 0.3 mM KCl) with 1.55 mg dithiotreitol/10 ml vol and 5 mg protease inhibitor/ml.

Samples were mixed on ice for 30 min, centrifuged at 12,000 x g at 4°C for 5 min, the supernatant was removed and the pellet discarded. Muscle extracts were mixed 1:1 with glycerol and stored at –20°C until analysed. Total protein was assayed using the Bradford total protein assay, with all samples measured in triplicate and the 2 closest samples averaged to give the protein concentration. The coefficient of variation (CV) of duplicate measurements of total protein content was 6.5%. Muscle extracts were diluted in sample buffer [400 mM Trizma Base, 1.8% (wt/vol) sodium dodecyl sulfate (SDS), 6.4% (vol/vol) glycerol, 4.0% (vol/vol) 2-mercaptoethanol, 8.8% (wt/vol) sucrose, 0.02% (wt/vol) bromophenol blue, pH 7.2] to give a final concentration of 0.15 μg/μl. Diluted muscle extracts were boiled for 5 min at 100°C, 20% iodoacetamide was added (10 μl/100 ml sample) and pH was monitored and maintained for 30 min at room temperature. Resultant standardized muscle extracts were stored at –20°C until analyzed electrophoretically. Pre- and post-training samples for each subject were prepared at the same time using the same solutions to ensure identical preparation.

Electrophoretic Analysis of MHC Isoforms

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis was performed to identify MHC isoforms. Separating gels were composed of 6.95% acrylamide, 0.139% bisacrylamide, 0.37 M Tris (pH 8.8), 35% glycerol, 0.1% SDS, 0.1% ammonium persulfate (APS) and 0.0375% TEMED, and the stacking gel consisted of 4.0% acrylamide, 0.08% bisacrylamide, 0.125 M Tris (pH 6.8), 25% glycerol, 0.1% SDS, 0.1% APS and 0.05% TEMED. Ten μl of standardized muscle extracts (1.5 μg/10 μl sample vol) were loaded in each lane and run for 24 hr at 10°C at 275 V using a Hoefer SE 600 electrophoresis system

(Amersham Biosciences, Montreal, Quebec). Running buffer consisted of 0.025 M Trizma Base, 0.01 M SDS and 1.44% glycine, with the upper chamber buffer also containing 24 mM 2-Mercaptoethanol. Gels were fixed for 30 min in 50% methanol, incubated for 30 min in a sensitizing solution of 50% glutaraldehyde, washed several times in distilled water, silver stained for 7 min in a solution of 0.1% NaOH, 1.15 % NH_4OH, 0.8% $AgNO_3$, followed by repeated washes with distilled water. The stained bands were revealed by a developing solution of 0.0024% citric acid, with this reaction stopped by a solution of 45% methanol and 10% glacial acetic acid. Gels were then scanned immediately.

Pre- and post-training samples were run in adjacent lanes to ensure identical run conditions, and gels were run in duplicate. A control sample of known MHC content was loaded on each gel. Bands corresponding to MHCI, MHCIIa and MHCIId(x), determined by known standards from our laboratory and published research, were identified. Gel documentation and densitometric evaluation of the relative content of each MHC was performed with SynGene Chemigenius imaging system and associated software (Synoptics, Cambridge, UK). Results are expressed as percentages of total MHC in each sample, with the values from both gels averaged. The CV for the relative MHC isoform content on duplicate gels was 3.9%, 3.3 %, and 10.4% for MHCI, IIa and IId(x), respectively.

Cortisol

Twenty-four hour urine was collected during the first and last week of training. Urine collection commenced 6 hrs post-training session and continued until 30 hr after the training session. During week 1, the collection period was following the second training session, and during week 9, the collection period was after the third training session of the week. The total volume of urine was measured and an aliquot was stored at –20 °C until analyzed. Urinary cortisol was measured using a commercially available kit (DiaSorin, Stillwater, MN). All samples were run in duplicate with an intra-assay CV of 8.2% for duplicate samples.

Dietary Monitoring

Dietary intake over a 3-4 day period was monitored every 2 weeks throughout the duration of the investigation. Subjects were provided with a checklist of the amount of protein in various foods and they attempted to maintain a daily protein intake of 1.5 g • kg body mass • day^{-1} (Lemon, 1994). Food intake, quantity and time ingested were recorded in booklets, with caloric and macronutrient intake determined using The Food Processor software (ESHA Research, Salem, OR).

Statistical Analysis

An independent sample Student's *t*-test was used to determine whether there was a difference between training groups in all baseline measurements. Separate two-factor analysis of variance (ANOVA) was used to determine whether there were differences between groups over time (repeated measure) for the dependent variables of strength, training variables (% of 1 RM, average repetitions per set, average load per set and training volume), cortisol response and protein intake. Separate three-factor ANOVA's were performed to analyze the effects of group and time with the variables of muscle fibre type area, proportions of muscle fibre type and MHC isoform proportions. A between factor of oral contraceptive use or non-use was initially included in all analyses but this factor did not demonstrate any significant main effects and thus the groups were collapsed across menstrual status. Statistical significance was set at $P \leq 0.05$ for all analyses and Newman-Keuls post-hoc procedures were used to locate differences when significant main effects or interactions were found. Results are reported as means and SD unless otherwise stated. All statistical analyses were carried out using the statistical software program Statistica 5.1 (StatSoft, Tulsa, OK).

Results

Subjects

Prior to training, there was no significant difference between groups in age, height, body mass, sum of six skinfolds (subscapular, tricep, iliac crest, abdomen, front and rear thigh), % body fat, sum of front and rear thigh skinfolds and body mass index (BMI). There was no significant change in body mass or BMI following training, and while there were changes in other variables (see Table 2), these changes did not differ (all $P > 0.05$) between groups. Mean (SD) menstrual cycle length with no oral contraceptive use during the cycle preceding commencement of training and over the training duration was 27.9 (2.2) days and 28.3 (1.3) days for LC (n=10) and LE (n=8), respectively, and did not differ between training groups.

Training Compliance

No subject missed more than 1 of 24 total training sessions. Of those subjects that did not complete a training session, 2 subjects were from LC and 5 subjects from LE. Training compliance for the 24 training sessions did not significantly differ between groups and was 99.4% (SD 1.5) and 98.4% (SD 2.1) of total training sessions for LC and LE, respectively. None of the subjects reported injury during the training period, although during the latter weeks of training, more subjects in the LC group reported general fatigue while more subjects in LE reported knee pain due to patellar tendonitis.

Strength Assessment

Leg Press

Table 3 shows the 1 RM values for concentric, eccentric and combined eccentric/concentric leg press strength at the 3 test periods (i.e. pre-, mid- and post-training). There were no significant differences in leg press maximal strength prior to training. There was a significant increase in 1 RM strength following training with significant main effects for time only, indicating that both groups experienced a similar increase in maximal strength.

For all subjects combined, 1 RM strength increases were: mean (SD); 21.3 (15.5)%, 25.2 (13.6)% and 43.6 (21.3)% for concentric, combined, and eccentric strength, respectively. The percentage increase in eccentric strength was significantly greater ($P \leq 0.01$) than the percentage increase of concentric and combined strength, which were similar in response.

Bench Press

The 1 RM values for concentric, eccentric and combined eccentric/concentric bench press strength at the 3 test periods (pre-, mid- and post-training) are shown in Table 4. There were no significant differences in bench press strength prior to training. There was a significant increase in 1 RM strength following training, main effects for time only, indicating that both groups experienced a similar increase in maximal bench press strength. For all subjects combined, 1 RM strength increases (mean, SD) were 14.6 (18.0)%, 9.8 (13.1)% and 28.2 (16.1)% for concentric, combined, and eccentric strength, respectively. The percentage increase in eccentric strength was significantly greater than the percentage increase of concentric and combined strength, which were similar.

Training Variables

Leg Press

The relative training load changed over the duration of training with no difference between LC and LE as indicated by a main effect for time. The relative training load increased over time such that there was a significant difference in relative training load between weeks 1, 5 and 9 (Table 5). The relative training load during all 3 occasions varied from 43-86% of combined 1 RM.

Leg press training variables are shown in Table 6. There were no significant differences between groups for the number of repetitions per set throughout the 9 weeks of training. As well, the number of repetitions per set did not significantly differ between weeks (mean 7 reps, SD 0.7 reps). The weekly average load per set did not significantly differ between groups and the load increased similarly for both groups. However, a significant main effect for time indicated that there was a gradual increase in load per set, with the load used during the last 4 weeks of training significantly greater than that used during the first 5 weeks of training. Likewise, total training volume increased ($P \leq 0.05$) over time, except for week 5 which consisted of only 1 training session. Thus the volume of leg press for weeks 6-9 was significantly greater than weeks 1-5.

Squat

Squat training variables are shown in Table 7. Analysis of average repetitions per set for squat showed no significant main effect for group or interaction of group by time indicating that both groups responded the same throughout the duration of training. However, a main effect for time revealed that for both training groups combined, there was a significant decrease in the number of repetitions per set for squat with training over time with a gradual decrease of 1 repetition per set over the 9 weeks. This decrease occurred mainly over the first 3 weeks of training, with the average number of repetitions per set of each of weeks 1-3 significantly greater than weeks 4-9. Regarding load per set, a significant main effect for time indicated that there was a gradual increase in load per set with significant increases in load occurring from week to week, with the exception of week 5-6 and week 7-8. There was also a significant group by time interaction for squat training load. Post hoc analysis revealed that while there was no significant difference between groups during weeks 1-8, during week 9, the average load of group LC was significantly greater than that of LE. As well, LC demonstrated a gradual increase in load over weeks with significant increases in load occurring between weeks 1-2, 2-3, 3-4, 6-7 and 8-9, while the increase in load over weeks for LE showed significant increases between subsequent weeks for week 1-2 and 2-3 only. Squat training volume demonstrated a significant increase (main effect for time) from week 1-2, 2-3, 5-6 and 6-7, with a plateau in volume over the last 3 weeks of training.

Leg Extension

Leg extension training variables are shown in Table 8. There were no significant differences between groups for the number of repetitions per set for leg extension throughout the 9 weeks of training. The mean (SD) repetitions per set for both groups was 6.9 (0.8). A significant main effect for time was observed, indicating that there was a gradual increase in load per set. The load during weeks 1 and 2 was less than the load in all subsequent weeks (all $P \leq 0.05$). The increase in average load was greater in the earlier weeks as indicated by a significant increase in load from week 1-2 and week 2–3, followed by a more gradual increase in load with a plateau in load during the last 3 weeks of training with no significant difference in load between weeks 7-9. The load used during the last 4 weeks of training was significantly greater than that used during the first 4 weeks of training.

Training volume for leg extension showed significant increases in training volume between weeks 1-2, 2-3, 3-4, and 5-6. The volume in the last 4 weeks of training was significantly greater than during the first 5 weeks of training. Training volume exhibited no significant differences in training volume between weeks 7-9.

Total Training Volume

Total training volume for the 3 leg exercises stressing the quadriceps muscle group (i.e.leg press, squat and leg extension) was also examined to determine if the interplay of the 3 exercises had an impact on volume (Table 9). There was no significant main effect for group or interaction of group and time for total training volume. However, total training volume during weeks 6-9 were significantly greater than weeks 1-3 and 5 (main effect time). Significant increases in total training volume between successive weeks occurred between weeks 1-2, 2-3, 3-4, 5-6 and 6-7 but not during weeks 7-9.

Fibre Type Cross-Sectional Areas and Proportions

Results are presented for the 26 subjects that had sufficient muscle tissue sample size for both pre- and post-training analyses (LC, n=14; LE, n=12; Table 9). Figure 1 indicates the different fire types on representative MHC immunohistochemical stains of serial cross-sections of the vastus lateralis muscle. CSA for mixed type-I/ IIA fibres and type-IID(X) fibres are not reported due to the low number of these fibres types with a mean of 2 mixed fibres and less than 1 type-IID(X) fibre per section. As well, the muscle biopsy samples of only 7 subjects contained mixed fibres both pre- and post- training and only 1 subject displayed type-IID(X) fibres pre- and post-training. No embryonic or type-IIB fibres were observed in any of the samples.

There was a significant 3-way interaction between group, time and fibre type area of type-I and -IIA fibres (Table 10). Both groups experienced a significant increase in the CSA of type-I fibres post-training while only the LC group demonstrated an increase ($P \leq 0.05$) in CSA of type-IIA fibres following training. A significant main effect for fibre type indicated that type-I fibres were larger ($P \leq 0.05$) than type-IIA fibres, collapsed across group and time. However, post hoc analysis of the 3-way interaction of group, time and fibre type area indicated that initially there were differences between groups in comparison of the size of each fibre type, and as a reflection of the different response of fibre types in terms of CSA to the different training protocols, each group had a different response. In the LC group, type-I fibres were larger ($P \leq 0.05$) than type-IIA fibres prior to training, but there was no difference in CSA post-training due to the greater increase in CSA of type-IIA fibres. The LE group demonstrated no difference in CSA of both fibre types initially, but type-I fibres were larger ($P \leq 0.05$) than type-IIA fibres following training.

There was a significant group by fibre type interaction for the percentage change in fibre type CSA with no significant main effects for group or fibre type. For the LC group, the relative change in muscle fibre CSA [mean (SD)] of type-I fibres [16.4 (7.2)%] and type-IIA fibres [25.8 (8.4)%] was similar for both fibre types ($P \geq 0.05$), while for the LE group, there was a significant difference in the percentage change of fibre types-I and -IIA [mean (SD)] [11.1 (4.4)% and –1.0 (4.1)%, for type-I and -IIA fibres, respectively]. Both groups demonstrated a similar increase in the percentage change in CSA of type-I fibres but the percentage change in size of type-IIA fibres was significantly different between groups.

Fibre type distribution pre- and post-training is shown in Table 11. No significant differences occurred in the proportions of each fibre type between groups prior to training. There was no difference between groups in the proportions of mixed type-I/IIA, type-IID/X and type-I fibres after training. The percentage of type-IIA fibres was greater ($P \leq 0.05$) in the LC group than the LE group following training. There was no change in the proportions of all fibre types following LC training. However, there was a shift in the proportions of type-I and -IIA fibre types with LE training, with an increase in the percentage of type-I fibres and a concomitant decrease in the percentage of type-IIA fibres (both $P \leq 0.05$). Both groups had a greater proportion of type-I fibres than type-IIA fibres pre- and post-training, and there were more type-IIA and -I fibres than mixed type-I/IIA and type-IID(X) fibres.

MHC Isoform Analyses

Representative MHC gels of a subject from each group are shown in Figure 2. There was no initial difference between groups in terms of the total proportions of each MHC isoform and no alterations in the proportion of each MHC isoform between groups occurred with training (see Table 12). A significant main effect for MHC isoform indicated that, collapsed over group and time, the proportion of MHCIId(x) was less than that of MHCIIa and MHCI, which did not significantly differ. A significant time by MHC isoform interaction revealed that both groups responded with a similar shift in MHC isoforms, with a significant decrease in the proportion MHCIId(x) accompanied by a significant increase in the proportion of MHCIIa. There was no significant change in the proportion of MHCI. The proportion of MHCIId(x) was significantly less than that of MHCIIa and MHCI both before and after training.

Cortisol

Values for 24-hr unextracted urinary cortisol levels during the first and last week of training are shown in Table 13. Urinary cortisol values did not initially differ between groups. However, a significant interaction of group by time showed that LC training produced a significant increase in 24-cortisol during week 9 while there was no change in 24-hr cortisol levels for LE training between weeks 1 and 9. All initial values were within the normal range of 75-270 μg per 24 hours, with the exception of one subject in LE, who had an initial value above the normal range. During week 9, this same subject showed levels above the normal range. As well, during week 9, 6 other subjects also had 24-hr cortisol levels above the normal range, 5 from LC and 1 from LE.

Dietary Intake

Macronutrient and caloric intake did not differ between groups during the 5 dietary recording periods and there was no alteration in macronutrient and caloric intake throughout the recording periods. For all recording periods, the mean (SD) percentage of total caloric intake for protein, carbohydrate and fat was 16 (5.0)%, 60 (8.4)% and 23 (7.2)%, respectively. Average daily caloric intake was 2280 (SD 418) kcals. Similarly, protein intake did not differ between groups during any of the dietary recording periods and did not change throughout the recording periods. Protein intake averaged 1.40 (SD 0.22) g · kg body mass · day^{-1}.

Discussion

There were four main findings observed in this investigation: (1) there was an equivocal response of both training groups in terms of increased strength (concentric, eccentric and combined eccentric-concentric strength); (2) preferential fibre type hypertrophy was induced with manipulation of concentric and eccentric muscle action time; (3) regardless of a differential response in fibre hypertrophy, a similar shift in MHC isoform content of both groups occurred with a decrease in the proportion of MHCIId(x) accompanied by an increase in the proportion of MHCIIa; (4) the changes that occurred were not attributable to differences in the training variables of repetitions per set, training load or volume.

There is a lack of previous investigations examining the role that varying TUT of concentric and eccentric muscle actions during DCER exercises has on subsequent adaptations. This is especially true when both actions utilize the same submaximal load, based upon concentric strength, and when the total time under concentric and eccentric tension is equated. As well, strength following DCER training is usually assessed by a combined eccentric/concentric action test only. This investigation found that both groups demonstrated significant and equivocal increases in strength, whether assessed concentrically or eccentrically only, or with a combined eccentric/concentric test. Thus, despite the difference in time to complete each muscle action, the inclusion of both concentric and eccentric muscle actions and the application of training resistance for both actions based upon the concentric load, there was no differential strength adaptations. These findings suggest that strength adaptations following DCER training with differences in time to complete each action, are general, and that the inclusion of both concentric and eccentric muscle actions during training are important to increase strength in all dynamic modes.

The finding of increased strength in all dynamic modes is supported by several studies that have examined strength responses following DCER training with either eccentric or eccentric actions only, both actions combined, or that have used the same concentric submaximal load used for both muscle actions (Ben-Sira et al., 1995; Dudley et al., 1991b; Häkkinen & Komi, 1981; Johnson et al., 1972). Training with either eccentric or concentric actions only has been reported to result in similar and significant increases in combined strength (Johnson et al.) or concentric only strength (Ben-Sira et al.). With time under tension matched between coupled eccentric and concentric actions or concentric only actions, Dudley and co-workers reported that both training protocols were effective at increasing combined and concentric only strength. Eccentric strength was not assessed in this latter study. There was some suggestion that the use of combined eccentric/concentric muscle actions during training was more effective than concentric action only training, as the combined action group demonstrating a trend of greater strength, with significantly greater strength increases in 1 of the 2 combined action tests. As well, the combined action training group also had a significantly greater increase in strength in one of the concentric only action tests compared to the concentric group that trained with the same TUT but with concentric actions only. This finding is also supported by the investigation of Häkkinen and Komi that included some eccentric actions during training resulting in significantly greater increases in combined action strength. While it is difficult to directly compare the results of the current investigation to above mentioned studies as the duration of eccentric and concentric muscle actions were not manipulated, the results of these investigations suggest that with DCER training, with equated muscle action time, repetitions, sets and volume, both eccentric or concentric muscle actions only result in increased concentric and eccentric strength. This would represent a non-mode specific increase in strength. As well, it may be necessary to include both eccentric and concentric muscle actions to maximize combined eccentric/concentric and concentric strength measurements. As well, when both actions are involved during training, test mode specificity may not be an issue.

There are a few studies that have manipulated concentric TUT as an individual training variable (Gillies & Docherty, 1999; Morrissey et al., 1998; Palmieri, 1989; Young & Bilby, 1993). In 3 of these studies, all muscle actions were completed in 2 s or less (Morrissey et al.; Palmieri; Young & Bilby) while Gillies and Docherty used 2 s to complete the eccentric actions and 2 and 8 s for the concentric actions. As well, training volume was also equated between groups. All of these investigations reported equivocal effects on combined and/or concentric strength. These results, along with the findings of the current investigation, suggest that concentric movement velocity during DCER training may not be important for increasing combined or concentric strength.

Overall, increases in maximal strength for all three modes tested varied from 21-44%, and were comparable to reports of increases ranging from 12-67% with investigations utilizing concentric and/or eccentric only and/or combined actions during training (Ben-Sira et al., 1995; Dudley et al., 1991b; Häkkinen & Komi, 1981; Johnson et al., 1972) or with manipulation of muscle action completion time (Gillies & Docherty, 1999; Morrissey et al., 1998; Palmieri, 1989; Young & Bilby, 1993). The use of "slow" muscle actions certainly did not detract from strength, as similar increases in combined or concentric strength result with other more "typical" protocols using combined action training (Bell et al., 2000; Carroll et al., 1998; Jürimäe et al., 1996; Staron et al., 1994; Williamson et al., 2001). The greatest change in leg press strength occurred with eccentric strength (44% increase), with smaller and similar increases of 25% and 21% for combined and concentric only strength, respectively. Bench press strength increases were more modest, likely due to the overall lower training volume for the upper body, and also showed a similar pattern of the greatest increase in eccentric strength (28%) with similar significant increases of 15% and 10% for combined and concentric 1 RM, respectively.

There is a lack of research that compares the differences in relative changes in eccentric strength with other modes of strength assessment, as most investigations have not assessed strength using all 3 modes. The observed greater change in eccentric strength may be partially explained by a greater potential to increase eccentric strength, as most individuals do not emphasize eccentric actions during resistance training or during daily activities. Increased strength during the early phases of resistance training programs is often attributed to neural mechanisms, especially with untrained individuals (Sale, 1988). During resistance training, there is often little emphasis on eccentric actions, and thus the requirement of control of eccentric movement velocity, which was novel for both training groups, may have resulted in neural adaptations and greater increases in eccentric strength in the present study. The relatively greater changes in eccentric strength may also be due to incomplete neuromuscular activation during maximal eccentric muscle actions (Aagaard et el., 2000; Amiridis et al., 1996; Enoka, 1996; Westing et al., 1990). This neuromuscular inhibition during eccentric actions has been reduced and muscle activation increased following DCER resistance training (Aagaard et el., 2000). While the exact mechanism is not known, the greater increase in eccentric strength may have been mediated through a reduction in an inhibitory neural drive/protective mechanism, which has been proposed to influence maximal force/torque production at slow velocities (Babault et al., 2002: Gulch, 1994; Linnamo et al., 2002; Perrine & Edgerton, 1978; Webber & Kriellers, 1997) and may be influenced with resistance training (Enoka, 1988; Jones et al., 1989; Moritani, 1992; Wilson, 1994).

The lack of significant differences in all strength assessments between groups may be explained by mechanical mechanisms and a greater ability of muscle to generate force eccentrically than concentrically (Donne & Luckwill, 1996; Gulling et al., 1996; Kellis & Baltzopoulos, 1998; Linnamo et al., 2002; Oskowski et al., 1995; Westing et al., 1991). Despite this difference in strength between concentric and eccentric muscle actions, the training load was determined by the concentric load, and may have limited potential strength adaptations. However, investigations that have used a greater eccentric than concentric load with a DCER mode, do not necessarily support that the use of greater eccentric than concentric loads results in enhanced strength (Ben-Sira et al., 1995; Carey Smith & Rutherford, 1995; Häkkinen & Komi, 1981; Johnson et al., 1976; Seliger et al., 1968). However, there is some support for the use of greater loads for the eccentric action to result in greater strength increases than equal loads for both muscle actions (Brandenburg & Dochery, 2002).

It is important to mention that the training protocols of the present study required a 1 s pause between muscle actions. This greatly reduced the effects of the stretch-shortening cycle (SSC) with the utilization of stored elastic energy (Cronin et al., 2001; Wilson, 1991) and increased muscle activation via the stretch reflex, resulting in an enhanced subsequent concentric action. The inclusion of a pause between muscle actions was desired during training as it minimized differences between subjects in their ability to use a SSC, and also reduced a tendency to increase velocity during the later part of the range of eccentric movement in anticipation of the concentric phase. The intent was to maintain an even speed of movement throughout both actions. Elimination of a SSC may have impacted upon the load used during training and potentially on the concentric and combined muscle action strength test.

During maximal strength assessment, many subjects demonstrated some difficulty with combined eccentric/concentric 1 RM attempts. This difficulty was offset by instruction to perform the test with "no pause after lowering" and to "explode up as hard and fast as possible" during the warm-up sets and during maximal attempts. Subjects were successful in eliminating a pause after several repetitions during the warm-up repetitions. Verbal encouragement was provide to all subject during 1 RM attempts. Recently, Toumi et al., (2001) reported that squat training with a pause or an isometric action between the eccentric and concentric phase was as effective in increasing isometric maximal force, and maximal force generated during a squat jump and countermovement jump as was training with rapid movement from eccentric to concentric phases. Heavy resistance training has also been found to not only enhance 1 RM combined strength but also improve force during SSC exercise (Wilson et al., 1996). These results indicate that the inclusion of a pause between muscle actions in the current investigation likely had minimal influence on concentric and combined maximal strength. It may be that the ability to use a SSC during training is more important for maximum power generation and rate of force development using much quicker movements with submaximal loads.

Preferential fibre type hypertrophy was induced with manipulation of concentric and eccentric muscle action time. The use of a slow concentric time during training resulted in significant hypertrophy of both type-I and -IIA fibres, which is similar to the findings of other resistance training investigations that have not manipulated TUT but have employed both concentric and eccentric muscle actions based on the same submaximal concentric load (Bell et al., 2000; Hather et al., 1991; Staron et al., 1991; Staron et al., 1989). When muscle fibre hypertrophy occurs, a significant increase in the CSA of type-II fibres (or subpopulations of type-II fibres) is not always accompanied with hypertrophy of type-I fibres (Kadi et al., 2000; Sharman et al., 2001), suggesting that hypertrophy of type-II fibres may precede that of type-I fibres. However, training with an emphasis on the eccentric action resulted in significant hypertrophy of type-I fibres only. To the author's knowledge, this is the first time that hypertrophy of only type-I fibres has been demonstrated following resistance training in humans with similar repetitions per set.

There are several explanations for this finding of differential hypertrophy with the training regimens. The first relates to muscle fibre recruitment. With the same submaximal load, EMG is lower during eccentric than concentric actions (Appendix A, Aura & Komi, 1986; Dalton & Stokes, 1991; Fang et al., 2001; McHugh et al., 2002; Nakazawa et al., 1992), suggesting that fewer motor units may be required during eccentric actions. This difference may be due to mechanical differences related to cross-bridge attachment/re-attachment and force production (Stauber, 1989) and thus lower muscle activation is required for eccentric actions. With an emphasis on the eccentric action during the LE protocol, the overall stimulus may not have been great enough to induce hypertrophy, and thus the smaller magnitude of increased muscle size observed in the present study with the LE protocol. Conversely, the longer TUT during the LC protocols may have optimized hypertrophy. Differences in motor unit behaviour, such as firing frequency and recruitment thresholds between concentric and eccentric actions have been previously reported (Howell et al., 1995; Nardone et al., 1989; Tax et al., 1989; Theeuwen et al., 1994). Thus these differences in neural activity and resulting greater muscle activation during concentric actions, and the longer TUT during the LC protocol, may have optimized hypertrophy of both fibre types.

There is some direct (Nardone et al., 1989) and indirect (Howell et al., 1995; Karapondo, et al., 1993; McHugh et al., 2002) evidence to suggest that there is preferential recruitment of type II motor units (fibres) during eccentric actions. This evidence led to the hypothesis (not substantiated) that there would be preferential hypertrophy of type-II fibres with LE, as they would receive greater activation and stimulation for hypertrophy. However, LE training induced hypertrophy of only type-I fibres, suggesting that this possible reversal of the size principle of motor unit recruitment did not occur, and type I motor units (fibres) had a greater involvement with the LE protocol and thus a greater stimulus leading to the finding of hypertrophy of type-I fibres only. During training, the repetitions were performed to failure, and most motor units should have been recruited. However, failure was determined by concentric, not eccentric failure, and combined with the emphasis on the eccentric action with the LE protocol, there may have been an overall greater stimulus to the type-I fibres with an insufficient stimulus to induce hypertrophy of type-II fibres. The shorter concentric TUT of the LE protocol may not have been a sufficient stimulus to induce hypertrophy of type-II fibres.

An alternate explanation for the differences in fibre type hypertrophy may be due to differences in muscle damage related to the two training protocols. The use of eccentric muscle actions, with greater associated loads than concentric actions, do cause muscle damage (Hortobagyi et al., 1998; Newham et al., 1983; Stauber, 1989; Waterman-Storer, 1991). As well, muscle damage is not only related to the greater load of eccentric actions, but is a result of the muscle action, with greater muscle damage occurring with eccentric than concentric actions using the same submaximal concentric load (Gibala et al., 1995), although muscle damage has been shown to be attenuated with prior resistance training experience (Gibala et al., 2000). Muscle damage is not limited to an acute exercise bout only, as muscle damage has been reported to be elevated following combined action resistance training with greater eccentric loads (Fiatarone Singh et al., 1999; Roth et al., 2000; Roth et al., 1999) or with the same load for both muscle actions (Staron et al., 1989; Staron et al., 1992). Several of these investigations also examined muscle hypertrophy and found that hypertrophy occurred along with evidence of muscle damage (Fiatarone Singh et al., Staron et al., 1989; Staron et al., 1992), suggesting that muscle fibre damage with subsequent regeneration is physiological signal initiating mechanistic cascades for muscle fibre hypertrophy. However, this is likely not the only signal as muscle damage has also been not observed following combined action resistance training in which hypertrophy occurred, as in the present study and also reported by Adams and co-investigators (1993).

The above mentioned investigations that observed muscle damage following resistance training did not specifically relate muscle damage to fibre type (Fiatarone Singh et al., 1999; Gibala et al., 1995; Gibala et al., 2000; Roth et al., 2000; Roth et al., 2000; Staron et al., 1989; Staron et al., 1992). There is some suggestion that fast type fibres are more susceptible to exercise-induced damage (Fridén et al., 1983; Lieber and Fridén, 1993; Macpherson et al., 1996). If preferential recruitment of type-II fibres occurred with the LE protocol, and muscle damage occurred throughout the training period, this could account for the lack of hypertrophy of type-II fibres. Conversely, if type-I fibres, with greater resistance to muscle damage, were more active with the LE protocol, this explanation could account for hypertrophy of type-I fibres. It may be that the LE protocol, with an emphasis on a long eccentric action, induced repeated muscle damage throughout the training period, irregardless of the type of muscle fibre recruited during the eccentric actions, and thus limited hypertrophy. The greater susceptibility to damage of type-II fibres could explain the lack of hypertrophy of this fibre type. While this explanation is contrary to the theory of muscle degeneration following by regeneration with subsequent hypertrophy of muscle fibres, it is possibly related to a greater amount of continual muscle damage with the LE protocol which thus limited an increase in fibre size. Hypertrophy of both fibre types with the LC protocol may be explained by the shorter TUT of the eccentric action, with potentially less muscle damage. While some muscle damage is expected, a lesser degree of muscle damage with the LC protocol, with an emphasis on the concentric action, may have been a sufficient stimulation for hypertrophy and account for the differential findings regarding fibre type hypertrophy between the LC and LE protocols.

The finding of no fibres with embryonic MHC was unexpected, as increases in fibres expressing embryonic MHC have been reported following resistance training (Fiatarone Singh et al., 1999; Kadi & Thornell, 1999) and suggest that muscle damage has occurred. These two investigations also provide additional evidence supporting degeneration and/or regeneration as a signal for hypertrophy. The increase in embryonic MHC post-training of the frail older adults in the investigation of Fiatarone Singh et al. (1999) was also accompanied by a large increase in damaged fibres, as well as significant type-II fibre hypertrophy, indicating on-going degeneration and regeneration accompanied with fibre hypertrophy. Increased embryonic MHC post-training in women reported by Kadi and Thornell (1999) was accompanied by a significant increase in satellite cells (Kadi & Thornell, 2000) along with preferential hypertrophy of type-IIa fibres (Kadi et al., 2000). While the increase in satellite cells suggests that muscle damage did occur, supporting degeneration and regeneration leading to hypertrophy, muscle damage was not assessed. An increase in satellite cell proportion, along with an increase in muscle damage following resistance training has been reported in young and older men and older women (Roth et al., 2000; Roth et al., 1999; Roth et al., 2001). However, increased satellite cell proportion was not accompanied with increased muscle damage in young adult women (Roth et al., 2000). While all groups demonstrated increased strength, muscle hypertrophy and myosin isoforms were not assessed. The effect of previous resistance training experience and habitual physical activity levels on the response of satellite cell activation and muscle hypertrophy requires investigation, as all subjects in the above mentioned studies had minimal resistance training experience and were sedentary. Further investigation is needed examining the relationship between training status, muscle damage, satellite cell activation, fibre regeneration and hypertrophy with different populations.

While there appears to be evidence in support of muscle degeneration followed by regeneration and hypertrophy, both the present study and the investigation of Adams and co-workers (1993) found no evidence of muscle damage but reported muscle fibre hypertrophy. Possible explanations for this difference in findings are: muscle damage is not a prerequisite for subsequent fibre hypertrophy; muscle damage is attenuated over the time course of training; or muscle damage may have occurred initially, followed by regeneration and subsequent resistance to injury (Devor & Faulkner, 1999). These latter 2 explanations may account for the lack of fibres expressing MHCembryonic in the present study. Another explanation is that the time between the final training session and that of the post-training biopsies may have allowed repair of damaged tissue. Gibala and co-workers (2000) found minimal evidence of muscle damage 5 days post-training session and thus the scheduling of the post-training muscle biopsy could influence the presence of damage. While muscle damage in the present investigation was examined on the basis of obvious morphological indication using light microscopy, which is limited in ability to detect microscopic damage, and the presence of embryonic MHC isoform, differences in muscle damage related to the training protocols may partially account for the finding of differences in fibre type hypertrophy with the different protocols and cannot be ruled out.

A third explanation regarding the differences in hypertrophy between LC and LE post-training could be due to alterations in connective tissue & cyotskeletal proteins [intermediate filaments (IF) and costameres] involved in the transmission of force from sarcomeres to whole muscle to the skeletal system (Enoka, 1998; Patel & Leiber, 1997; Rutherford, et al., 1989). It has been demonstrated that chronic low-frequency electrical stimulation can induce increased expression of IF proteins (Baldi & Reiser, 1995). Alteration in some of these proteins as a result of the LE protocols, with an emphasis on the eccentric action and fewer motor unit involvement, could lead to increased force transmission and thus a hypertrophic response of contractile proteins was not needed to meet the demands imposed by training. This mechanism remains speculative.

Another potential mechanism regarding differential hypertrophy of the different training protocols could be due to a difference in synergist muscle activation during concentric and eccentric muscle actions. While muscle activation of the quadriceps muscle group was not examined in the present study, previous investigations have found an alteration in synergist muscle activation dependent on muscle action (Appendix A; Nakazawa et al., 1993; Narici, et al., 1996: Tax et al., 1990). Differences in the relative involvement of the muscles of the quadriceps muscle group could have occurred with the different training protocols, with emphasis on different muscle actions. While hypertrophy of the vastus lateralis (VL) was limited with LE, increased thigh girth with decreased skinfolds thickness suggests that there was an overall increase in thigh muscle volume that may not have been reflected by alteration in CSA of the VL only. Narici and co-workers found that the rectus femoris (RF) was the most active of the quadriceps muscle during the eccentric phase of leg extension. Thus the differences in hypertrophy between LC and LE may be partly a result of differences in muscle activation of the synergist muscles related to an emphasis on the concentric or eccentric action. As well, the limitations of a single biopsy site, along with the additional consideration that the VL was one of several muscles involved with the training exercises do not infer that similar changes occurred throughout the muscle group.

Differences in metabolic stress with the 2 training protocols could also account for the differences in hypertrophy between LC and LE. There is a greater metabolic demand during concentric actions than during eccentric actions (Dudley et al, 1991a: Ryschon et al., 1997), thus the metabolic demand of LC, with the emphasis on the concentric action, was likely greater than that of LE. While not directly examined, more subjects in LC noted general fatigue, which could be linked to greater metabolic requirements of LC. Both groups had adequate protein intake throughout the duration of training and caloric intake remained unaltered, thus protein and energy intake were not confounding factors.

Indirect evidence of greater metabolic stress with LC may be provided by the significant increase in 24-hr urinary cortisol of LC. As well, during the final week of training, 5 of 7 subjects with 24-hr cortisol values above the normal range were from the LC group. While it would be expected that increased cortisol levels with the associated catabolic effects on muscle tissue would limit muscle hypertrophy, LC experienced significant hypertrophy of both fibre types, suggesting that the LC training protocols were very effective at stimulating muscle growth despite the catabolic effects of cortisol. This finding of increased cortisol levels in LC is not in agreement with previous research that has found no increase in cortisol levels in women following resistance training with TUT not controlled or specified (Bell et al., 1997; Bell et al., 2000; Staron et al., 1994), while no change in cortisol with LE is in agreement with these prior investigations. This difference suggests that in the present study, a greater metabolic stress occurred with the LC than the LE protocol. The stimulus for hypertrophy with LC training, despite the catabolic effects of cortisol, could be related to other associated anabolic hormone and growth factors, such as testosterone, human growth hormone (hGH) or insulin-like growth factor I. While Bell and co-investigators (2000) found no change in testosterone or hGH in women following resistance training, other anabolic hormones may be implicated. As well, different signaling pathways may be stimulated as a result of by-products related to anaerobic metabolism, such as lactic acid, inorganic phosphate or creatine. Other factors such as muscle pH, muscle temperature, alterations in calcium homeostasis and satellite cell activation may also be implicated in the hypertrophic response to resistance training. These factors have not yet been adequately examined with concentric and eccentric muscle actions, or with actions of different durations. Thus it is possible that the differential response in hypertrophy between the training groups may be related to different physiological signals.

While fibre type transitions following resistance training usually occur within the fast fibre type populations such that there is an decrease in the proportion of type-IIB fibres with a concomitant increase in the proportions of type-IIA (Adams et al. 1993; Hather et al., 1991; Staron et al., 1989; Staron et al., 1991; Staron et al., 1994), this pattern was not observed with either LC or LE. However, the present result of no change in the proportions of fibre types in LC is in agreement with Chilibeck and co-investigators (1999), and may be related to the significant hypertrophy of both type-I and -II fibre types in both of these investigations. Alterations in the proportion of type-I fibres following resistance training have not been noted, with the exception of Kadi and co-workers (2000). In that investigation, there was a significant decrease in the proportion of type-I fibres along with an increase in type-IIA fibres. However, this result supports the results of other investigations (Adams et al. 1993; Hather et al., 1991; Staron et al., 1989; Staron et al., 1991; Staron et al., 1994; Williamson, et al., 2001) that suggest that the end-point of fibre type conversion with resistance training is toward type-IIA. The increase in the proportion of type-I fibres with a parallel decrease in type-IIA fibres with LE training in the present study is unique and contrary to the direction of fibre type transitions usually reported following resistance training. However, this shift toward slower fibre phenotype did not affect functional strength, while the effect on other muscle properties is unknown.

Despite a differential response in fibre hypertrophy, the present results for both LC and LE of a decrease in the proportion of MHCIId(x) with a concomitant increase in the proportion of MHCIIa illustrate a significant training induced shift in fast MHC isoform content that is in agreement with previous resistance training studies that have noted a similar shift with no alteration of MHCI isoform content (Adams et al., 1993; Carroll et al., 1998; Jürimäe et al., 1996; Sharman et al., 2001; Williamson et al., 2001). While Kadi and Thornell (1999) did report an alteration in MHCI, there was a bi-directional shift in MHC proportions toward MHCIIa, with a decrease in both MHCI and MHCIId(x). Together, the findings of these investigations as well as the current investigation all support that the shift in MHC content following resistance training is towards increased MHCIIa. There is some suggestion that this alteration in MHC content is related to a reduction in hybrid fibres that co-express multiple MHC isoforms, resulting in an increase in the proportion of fibres expressing MHCIIa only (Williamson et al., 2001). However, it must also be considered that the observed shift in MHC isoforms may not represent a true shift in proportions but may be a reflection of relative fibre type hypertrophy.

There appeared to be a mismatch between the changes in fibre type proportion and MHC content in LE. This finding is not in agreement with the few investigations that have examined both fibre phenotype and MHC expression (Adams et al., 1993; Hather et al., 1991; Kadi et al., 2000; Kadi & Thornell, 1999) as in these studies, the change in fibre type proportions mirrored the change in MHC content. An increase in the number of fibres co-expressing MHCI as well as some MHCIIa could account for this mismatch, but there was no change in the number of hybrid type-I/IIA fibres. As there were more type-I fibres and they became larger with LE, it is possible that a slight increase in MHCIIa expression in these fibres was not great enough to change classification from type-I to mixed, but this alteration was detected by MHC quantification. However, the findings of Williamson and co-workers (2001) of a reduction in fibres expressing more than one MHC following resistance training suggest that increased multiple MHC co-expression would not have occurred. The area of type-I fibres was greater than that of type-IIA fibres before LC training but there was no difference in area following training. Thus, the increase in the proportion of MHCIIa after training may be a reflection the greater relative increase in the size of type-IIA fibres, and not just a shift in the proportion of the fast MHC's. However, the same explanation cannot be used for LE, as there was no initial difference in fibre type area prior to training and type-I fibres were larger than type-IIA fibres following LE training. This would suggest that the proportion of MHCI would also increase, but this was not found. This difference in agreement between proportions of fibre phenotype and MHC content in the present study may be due to the training protocols of LE or the previously trained status of subjects.

The present results also suggest that resistance training influences mainly the fast MHC isoforms, or that the end-point of fibre type transformations following resistance training is towards increased MHCIIa. Another possibility for this mismatch of fibre phenotype and MHC expression with LE may be that with continued training, hypertrophy of type-IIA fibres might have occurred, as MHC transformation in muscle fibres has been shown to precede hypertrophy (Staron et al., 1991). Conversely, there was a slight (although not significant) increase of MHCI in LE that with a longer training period, a transformation towards MHCI might occur and match the alteration in fibre phenotype.

The present results strongly suggest that changes observed were due to the manipulation of the time to complete the concentric and eccentric muscle actions and not due to acute program variables. There were no differences between LC and LE for any training variable for the 3 exercises (leg press, parallel squat and leg extension) involving the quadriceps muscle group and the VL (leg press, parallel squat and leg extension) in load per set, repetitions per set or training volume throughout the training period. The only exception was LC using a greater load for squat during the last week of training. It is not likely that this difference in training load for 1 of the 3 exercises stressing the quadriceps muscle group during the last week of training can account for the observed differences if fibre type hypertrophy between LC and LE.

The training protocols of the current investigation required subjects to use a load that elicited technical failure within 6-8 repetitions, which resulted in a leg press load that did not differ between groups and varied from 56-67% of 1 RM over the duration of the current study. This % of 1 RM is lower than what would be expected with the given repetitions. Hoeger and co-workers (1990) reported that resistance-trained females were able to complete 57 and 24 repetitions of leg press with a load of 60 and 80% of 1 RM, respectively, using an unspecified cadence with no pause between repetitions. While the leg press exercise of this investigation differed from that of the current investigation, other resistance training exercises were also examined. The findings illustrate that the protocols of the current investigation requiring a relatively slow average velocity of the eccentric or concentric muscle actions resulted in a reduced training load. It should also be noted that while the mean training load was reduced, there was considerable variation in the relative load used by individual subjects (varied from 43-86% of 1 RM). Thus the current results regarding the relative training load, and the increase in relative training load throughout the training duration, as well as the range in individual relative training loads, suggests that training protocols that use a training load determined as a set percentage of 1 RM, even if 1 RM is re-tested and the load is re-set throughout the training period, may not be optimal for all subjects and may compromise the resultant findings. As well, the lower training load of the current study emphasizes that it is not the load only that influences muscle hypertrophy, but how the load is applied.

In conclusion, the present investigation has shown that preferential fibre type hypertrophy can occur with the manipulation of the time to complete the concentric or eccentric muscle actions when the same sub-maximal concentric load is used for both muscle actions. An emphasis on the concentric action appears to optimize fibre type hypertrophy while an emphasis on the eccentric action results in minimal hypertrophy related to only type-I fibres. These differences in hypertrophy were not accompanied by a differential response in strength, MHC protein expression or differences in training variables, and may be related to metabolic factors due to an emphasis on the specific muscle action.

Table 1. Experimental design and assessment schedule.

Week	0			1	5	9	10	
Day of Week	1	3	5	5	5	5	2	5
Anthropometry	X							X
Muscle biopsy	X							X
1 RM strength		X			X		X	
Exercise Familiarization			X					
24-hr urine collection				X		X		

Table 2. Anthropometric measurements and body composition pre- and post-training.

	Long Concentric n= 15		Long Eccentric n=13	
Variable	Pre	Post	Pre	Post
Body Mass (kg)	65.4 (8.3)	65.2 (6.9)	68.1 (10.6)	68.9 (10.4)
Sum of 6 skinfolds (mm)	90.9 (19.3)	86.7 (12.8)*	105.9 (23.2)	100.4 (18.2)*
Thigh skinfolds [sum of front & rear thigh (mm)]	38.4 (12.3)	34.1 (7.3)*	41.5 (8.6)	38.1 (6.2)*
Mid-thigh girth (cm)	49.4 (2.5)	49.8 (2.5)*	50.7 (3.2)	51.3 (2.7)*
Body fat (%)[1]	16.2 (5.0)	15.1 (3.1)*	19.2 (5.5)	18.0 (4.3)*
BMI (kg • m^{-2})	23.9 (3.1)	23.8 (2.6)	25.6 (4.2)	25.8 (4.1)

All values are means and SD.

* indicates significant ($P \leq 0.05$) difference from previous measurement.

[1] determined by prediction equation using sum of 6 skinfolds (Yuhasz, 1966).

Table 3. Leg press maximum strength (1 RM) for the three strength tests at pre-, mid- and post-training.

	Concentric			Combined			Eccentric		
Group	Pre	Mid	Post	Pre	Mid	Post	Pre	Mid	Post
LC	253.6 (52.4)	283.3 (54.8)*	302.1 (62.3)*	289.8 (58.0)	331.1 (68.4)*	354.1 (76.3)*	308.0 (67.5)	368.6 (70.0)*	417.6 (84.6)*
LE	271.0 (55.2)	319.7 (449)*	326.0 (41.3)*	297.9 (59.8)	363.8 (78.3)*	378.1 (63.0)*	317.1 (68.0)	406.6 (62.7)*	467.8 (59.1)*

All values in kg [mean (SD)]. LC (Long Concentric), LE (Long Eccentric).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

Table 4. Bench press maximum strength (1 RM) for the three strength tests at pre-, mid- and post-training.

	Concentric			Combined			Eccentric		
Group	Pre	Mid	Post	Pre	Mid	Post	Pre	Mid	Post
LC	42.3 (9.7)	43.9 (9.5)	48.0 (11.6)*	45.9 (10.6)	46.8 (10.9)	49.4 (9.0)*	51.5 (10.5)	57.9 (10.5)*	66.2 (11.9)*
LE	41.1 (10.5)	42.0 (7.0)	42.0 (5.5)*	44.9 (10.0)	44.8 (8.0)	48.5 (6.3)*	52.1 (11.5)	57.3 (9.1)*	64.1 (5.0)*

All values in kg [mean (SD)]. LC (Long Concentric), LE (Long Eccentric).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

Table 5. Relative training load (percentage of maximal combined 1 RM leg press strength) during weeks 1, 5 and 9.

Group	Week 1	Week 5	Week 9
LC	54 (6.6) [43-66]	62 (9.1)* [47-77]	68 (12.3)* [47-85]
LE	58 (8.3) [47-73]	60 (11.12)* [43-78]	66 (11.1)* [45-86]

All values are mean % 1 RM (SD) and [range]. LC (Long Concentric), LE (Long Eccentric).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

Note that differences between groups were not significant.

Table 6. Training variables of weekly mean repetitions per set, mean load per set and total volume (sum of repetitions x load for each set) for leg press throughout the 9 training weeks.

		Week								
Group	Variable	1	2	3	4	5	6	7	8	9
LC	Repetitions (#)	7.1 (0.7)	7.0 (0.8)	6.9 (1.0)	7.1 (0.7)	6.3 (1.8)	7.0 (0.5)	6.9 (0.5)	7.0 (0.6)	7.0 (0.7)
	Load (kg)	150 (19)	162 (18)	176 (23)	190 (27)	186 (60)	205 (28)+*	215 (28)*	224 (30)*	233 (30)*
	Volume (kg)	4385 (915)	7117 (1731)+	7602 (2045)	8396 (2023)+	2635 (1078)+	8611 (2050) +*	9142 (1639)*	9509 (2412)*	9352 (3222)*
LE	Repetitions (#)	7.4 (0.6)	7.2 (0.5)	6.6 (2.0)	6.6 (2.1)	6.7 (2.1)	7.1 (0.5)	7.1 (0.7)	6.8 (0.7)	6.3 (2.0)
	Load (kg)	166 (22)	181 (33)	182 (67)	193 (76)	198 (79)	223 (49)+*	229 (49)*	239 (48)*	225 (82)*
	Volume (kg)	4810 (874)	7540 (1813)+	7702 (3046)	8615 (4641)+	3025 (1581)+	9524 (1956)+*	10233 (3990)*	9913 (3481)*	8165 (4319)*

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric). Repetitions are the mean number of repetitions during all sets of each week. Training load was determined as the mean load utilized per repetition during all sets of each week. Training volume for each week was calculated by the sum of load x repetitions for each set. Note that there were no significant differences between groups at each week for all variables. + indicates significant ($P \leq 0.05$) difference from previous week. * indicates significant ($P \leq 0.05$) difference from weeks 1-5. Note that there was 1 training session only during week 5.

Table 7. Training variables of weekly mean repetitions per set, mean load per set and total volume (sum of repetitions x load for each set) for squat exercise throughout the 9 training weeks.

		Week								
Group	Variable	1	2	3	4	5	6	7	8	9
LC	Repetitions (#)	7.6 (0.7)	7.2 (0.8) +	7.1 (0.6)	7.0 (0.7)+	6.9 (0.8)	6.7 (0.8)	6.9 (0.6)	6.6 (0.9)+	6.7 (0.6)
	Load (kg)	28 (6)	34 (8)+	41 (11)+	47 (12)+	50 (11)	50 (13)	55 (12)+	59 (13)+	62 (15)+
	Volume (kg)	868 (238)	1518 (570)+	1815 (599)+	2036 (745)	685 (297)+	2091 (758)+	2231 (748)+*	2374(863)*	2451 (798)*
LE	Repetitions (#)	7.8 (0.4)	7.5 (0.4)+	7.6 (0.7)	6.9 (0.5)+	7.2 (0.7)	6.9 (0.9)	7.1 (0.7)	6.8 (0.7)+	6.9 (0.7)
	Load (kg)	26 (7)	33 (10)+	41 (11)+	46 (10)+	47 (11)	50 (11)	51 (12)	53 (12)	54 (13)
	Volume (kg)	815 (232)	1427 (512)+	1801 (422)+	1876 (819)	674 (348)+	1947 (429)+	2198 (979)+*	2137 (867)*	1988 (530)*

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric). Repetitions are the mean number of repetitions during all sets of each week. Training load was determined as the mean load utilized per repetition during all sets of each week. Training volume for each week was calculated by the sum of load x repetitions for each set. Note that there were no significant differences between groups at each week for all variables except for the average training load during week 9. + indicates significant ($P \leq 0.05$) difference from previous week. * indicates significant ($P \leq 0.05$) difference from weeks 1-5. Note that there was 1 training session only during week 5.

Table 8. Training variables of weekly mean repetitions per set, mean load per set and total volume (sum of repetitions x load for each set) for leg extension throughout the 9 training weeks.

		Week								
Group	Variable	1	2	3	4	5	6	7	8	9
LC	Repetitions (#)	6.9 (0.8)	7.1 (0.8)	7.0 (0.6)	6.8 (0.5)	6.8 (2.5)	6.7 (0.4)	6.7 (0.6)	6.9 (0.9)	6.7 (0.5)
	Load (kg)	17 (5)	21 (4)+	26 (6)+	30 (5)+	31 (11)	33 (7)+*	36 (6)*	38 (8)*	40 (9)*
	Volume (kg)	487 (166)	906 (196)+	1027 (275)+	1213 (223)+	449 (175)+	1344 (245)+*	1438 (249)*	1594 (419)*	1538 (384)*
LE	Repetitions (#)	7.4 (0.3)	7.1 (0.7)	7.4 (0.4)	7.2 (0.7)	6.5 (2.3)	7.0 (0.5)	7.0 (0.7)	7.0 (0.6)	6.8 (0.5)
	Load (kg)	20 (5)	25 (7)+	28 (7)+	32 (8)+	30 (13)	35 (8)+*	37 (9)*	39 (9)*	40 (9)*
	Volume (kg)	593 (158)	1050 (345)+	1281 (335)+	1321 (378)+	438 (201)+	1457 (403)+*	1564 (427)*	1585 (471)*	1573 (517)*

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric). Repetitions are the mean number of repetitions during all sets of each week. Training load was determined as the mean load utilized per repetition during all sets of each week. Training volume for each week was calculated by the sum of load x repetitions for each set. Note that there were no significant differences between groups at each week for all variables. . + indicates significant ($P \leq 0.05$) difference from previous week. * indicates significant ($P \leq 0.05$) difference from weeks 1-5. Note that there was 1 training session only during week 5.

Table 9. Total training volume (weekly sum of repetitions x load for each set of leg press, squat and leg extension) throughout the 9 training weeks.

		Week								
Group	Variable	1	2	3	4	5	6	7	8	9
LC	Volume (kg)	5722 (1202)	9481 (2129)+	10376 (2349)+	11564 (2537)+	3740 (1203)+	11956 (2433)+*	12715 (2093)*	13371 (3037)*	13239 (3832)*
LE	Volume (kg)	6250 (1261)	10150 (2619)+	11057 (3341)+	11845 (4912)+	4102 (1882)+	12817 (1954)+*	13878 (4460)*	13502 (4089)*	11598 (4457)*

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric). Note that there were no significant differences between groups at each week for all variables. Training volume for each week was calculated by the sum of load x repetitions for each set of leg press, squat and leg extension. Note that there were no significant differences between groups at each week for all variables. + indicates significant ($P \leq 0.05$) difference from previous week. * indicates significant ($P \leq 0.05$) difference from weeks 1-5. Note that there was 1 training session only during week 5.

Table 10. Muscle fibre cross-sectional area (μm^2) pre- and post-training.

	Pre Training		Post Training	
	Type-I	Type II-A	Type- I	Type-IIA
LC	3336 (470) #	2950 (486)	3833 (741)*	3676 (882)*
LE	3843 (1236)	3695 (598)	4239 (1392)* #	3633 (604)

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

# indicates significant ($P \leq 0.05$) difference between fibre type areas of the same biopsy (same time of sampling) within a group.

Table 11. Muscle fibre type proportions (%) pre- and post-training.

	Pre Training				Post Training			
	Type-I	Type-IIA	Mixed	Type-IID(X)	Type-I	Type-IIA	Mixed	Type-IID(X)
LC	55.3 (9.6)	43.2 (8.4)	1.2 (3.9)	0.3 (1.2)	54.9 (9.4)	44.6 (9.6)#	0.5 (1.0)	0.0 (0.1)
LE	55.1 (13.9)	44.5 (13.7)	0.4 (0.7)	0.1 (0.3)	60.6 (14.5)*	38.9 (13.8)*	0.5 (1.1)	0.0 (0.2)

All values are mean (SD). LC (Long Concentric), LE (Long Eccentric). Mixed fibres are type-I/IIA.

* indicates significant ($P \leq 0.05$) difference from previous measurement.

# indicates significant ($P \leq 0.05$) difference between groups.

Table 12. Myosin heavy chain composition (% of total MHC) pre- and post-training.

	Pre Training			Post Training		
	MHCI	MHCIIa	MHCIId(x)	MHCI	MHCIIa	MHCIId(x)
LC	45.2 (7.6)	44.9 (5.6)	10.0 (5.7)	43.0 (6.7)	48.9 (5.7)*	8.1 (3.8)*
LE	47.0 (8.3)	43.8 (4.5)	9.3 (6.7)	47.4 (7.5)	47.3 (7.6)*	5.4 (4.6)*

All values are mean percentage (SD). LC (Long Concentric), LE (Long Eccentric).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

Table 13. Twenty-four hour urinary cortisol (μg per 24 hours) during weeks 1 and 9.

	Week 1	Week 9
LC	181.2 (53.9)	240.0 (99.1)* #
LE	187.7 (58.2)	187.0 (80.5)

All values are mean (SD).

* indicates significant ($P \leq 0.05$) difference from previous measurement.

# indicates significant ($P \leq 0.05$) difference between groups.

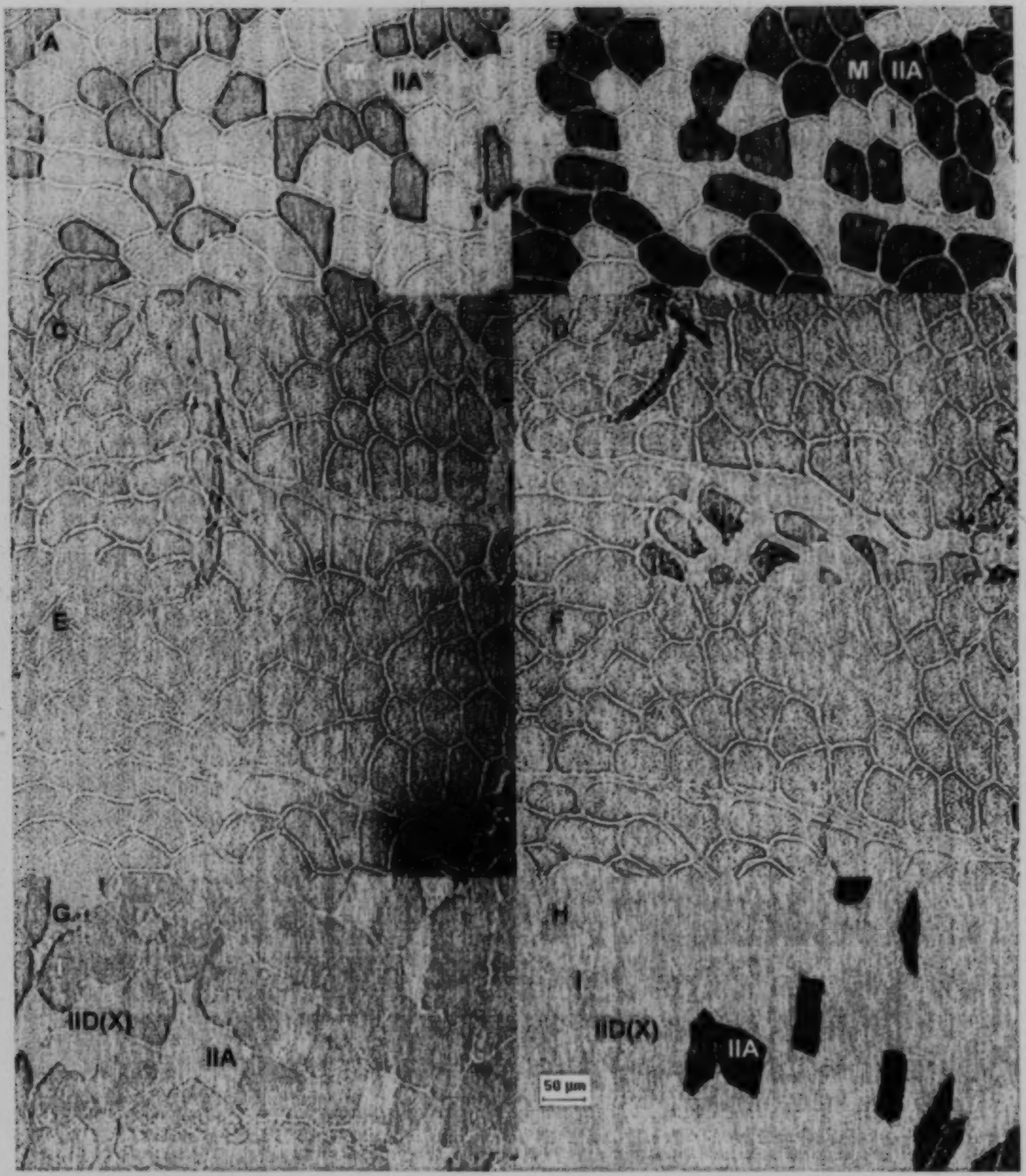
A
M
IIA
B
M
IIA
I
C
D
E
F
G
I
IID(X)
IIA
H
I
IID(X)
IIA
50 µm

Figure 1. Representative MHC immunohistochemical stains of serial cross-sections of the vastus lateralis muscle.

Sections are stained for: MHCI with monoclonal antibody (mAB) clone BA-D5 (A and G); for MHCIIa with mAB clone SC-71 (B and H); for MHCembryonic with mAB clone BF-45 (C); MHCIIb with mAB clone BF-F3 (D); IgG control (E); IgM control (F). One typical fibre of each type is labeled (I, IIA, Mixed I & IIA-M, IID(X).

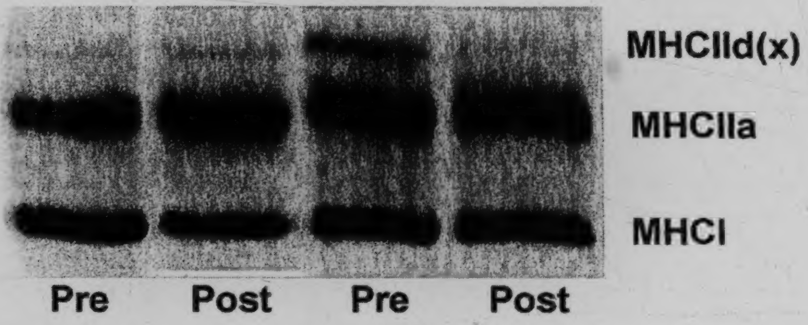
MHCIId(x)
MHCIIa
MHCI
Pre
Post
Pre
Post

Figure 2. Representative gels showing MHC isoforms separated by SDS-PAGE. Bands are shown from 2 subjects before (Pre) and after (Post) 9 weeks of resistance training. Left 2 lanes are from a LC subject and right 2 lanes are from a LE subject.

CHAPTER III GENERAL DISCUSSION AND CONCLUSIONS

The most interesting finding of the current investigation with manipulation of the time to complete the concentric and eccentric muscle actions during DCER resistance training using the same submaximal concentric load for both actions was related to selective hypertrophy of fibre types. The use of a longer time to complete the concentric action (lower average velocity) maximized hypertrophy of both type-I and -IIA fibre types, while the use of a longer time to perform the eccentric action resulted in a much smaller change in muscle size with hypertrophy of type-I fibres only. It was also interesting that this differential response in fibre type hypertrophy was accompanied by equivalent increases in all strength assessments (eccentric, concentric and combined eccentric/concentric strength) and MHC alterations, and was not due to differences in training variables.

Examination of the neural response of eccentric and concentric muscle actions with the same submaximal load and with a range of velocities (or time to complete the muscle action) is limited. Muscle activation has been examined more often with an isokinetic mode and the associated maximal force generation throughout the ROM (Donne & Luckwill, 1996; Gulling et al., 1996; Kellis & Baltzopoulos, 1998; Komi et al., 2000; Westing et al., 1991), as opposed to a DCER mode using a submaximal load. The results of a study reported in Appendix A demonstrate that with the same submaximal load, neural activation is lower during eccentric as opposed to concentric muscle actions, indicating that fewer motor units and muscle fibres are involved with eccentric actions. While motor type behaviour was not specifically examined, the results of Appendix A support the notion that faster motor units may be involved with eccentric muscle actions, as suggested by others (McHugh et al., 2002; Nardone et al., 1989).

The current investigation attempted to match the total neural activation of both eccentric and concentric muscle actions (resulting in the chosen times to complete the muscle actions of both LC and LE of the current investigation). Thus adaptations following training could be related to preferential recruitment of fibre types dependant upon emphasis on concentric or eccentric muscle actions. The results of the current investigation regarding muscle fibre hypertrophy do not suggest that a difference in fibre type recruitment occurs with eccentric and concentric muscle actions. The hypertrophy of both type-I and -IIA fibres following LC training was expected, and suggests that the emphasis on the concentric action provided a stimulus to both fibre types. The LE training protocols, with an emphasis on slow eccentric actions, increased fibre size of type-I fibres only, suggesting that type-I fibres were primarily involved.

However, this differential response in fibre type hypertrophy may not only be due to fibre type activation of eccentric and concentric muscle actions, but may be due to differences in muscle damage related to the training protocols. Greater muscle damage has been associated with eccentric actions (Gibala et al., 1995, 2000; Hortobagyi et al., 1998; Newham et al., 1983; Stauber, 1989; Waterman-Storer, 1991). If muscle damage was greater with the LE protocol and occurred throughout the training duration, this could account for the lower degree of fibre type hypertrophy found with LE.

The differences between the LC and LE training protocols may be related to the kinematic and kinetic patterns of different times to complete the muscle action (average velocity). Specifically, the lack of differences in kinematic and kinetic responses between similar eccentric and concentric velocities of the prior investigation (Appendix A), as well as an even level of force throughout the slowest velocity movements, combined with the similar training responses of the current investigation, further supports the suggestion that the differential finding regarding fibre type hypertrophy of the LC and LE training protocols was related to differences in fibre type recruitment.

The use of either LC or LE protocols may be useful to different populations, depending on the desired outcomes of a resistance training program. In view of the significant hypertrophy of both fibre types of LC, the use of the LC protocols is obviously applicable to individuals that wish to maximize increases in muscle size and strength. Conversely, the LE protocols, using submaximal loading based on the concentric action, would be appropriate for individuals that want to increase strength but do not want increased muscle size. This is especially important for sports with body mass categories or where increased body mass or muscle size could be detrimental to performance. A similar finding of strength increases with minimal hypertrophy following DCER training using submaximal eccentric only actions was reported by Housh and co-investigators (1998a). The same investigators (Housh et al., 1998b) also found that with the same training variables, but with concentric only actions, both strength and hypertrophy were maximized. Interestingly, it has been recently reported that velocity during eccentric actions may effect the hypertrophic response, with greater hypertrophy following fast, as opposed to slow eccentric training, as well as fast and slow concentric only training (Farthing & Chilibeck, 2001). While these findings appear to contradict the current results, this differential finding regarding hypertrophy may be due to differences in training load, as Farthing and Chilibeck used an isokinetic mode which results in maximal force generation throughout the ROM, as opposed to the submaximal loads with the DCER training of the current study. DCER investigations using an augmented load for the eccentric actions have also not found greater hypertrophy with the use of a higher load during the eccentric actions (Brandenburg & Docherty, 2002; Carey Smith & Rutherford: 1995).

It is clear that maximal eccentric exercise induces severe muscle disruption and damage (Hortobagyi et al., 1998; Newham et al., 1983; Stauber, 1989; Waterman-Storer, 1991). Muscle damage is not limited to only maximal eccentric forces, but can also occur following submaximal (80% of concentric 1 RM) eccentric, as well as concentric actions, although damage is attenuated with concentric actions (Gibala et al., 1995, 2000). The load used with the LC and LE protocols was much lower than more "typical" protocols, which could further reduce muscle damage. As well, if preferential recruitment of type-I fibres occurred with LE, or if type-I fibres were more active with LE, this suggests that the use of LE protocols may also be applicable/ desired in order to minimize the effects of muscle damage, as type-II fibres are more susceptible to exercise-induced damage (Fridén et al., 1983; Lieber and Fridén, 1993; Macpherson et al., 1996). While not documented, none of the subjects reported any acute effects such as short-term loss in strength, pain, stiffness or decreased ROM with the commencement of training. Although muscle damage is not necessarily detrimental to adaptations following resistance training, age and gender may influence susceptibility to exercise-induced muscle damage. Muscle damage has been shown to increase in elderly women following resistance training, while older men and younger women and men did not show increased muscle damage (Roth et al., 2000; 1999), suggesting that the LE protocol may be appropriate for elderly women.

The use of slow eccentric actions during resistance training may also be applicable to other populations. Resistance training using the LE and LC protocols could aid in fall prevention and recovery. The use of controlled muscle actions with both protocols could increase familiarity with eccentric actions, as well as the ability to exert relatively high levels of force over a longer period of time than that required for most activities of daily living. Thus familiarity with eccentric actions and the ability to generate high levels of force for a number of seconds, and not just a quick powerful movement, could be important for prevention or recovery from a fall.

Eccentric actions are associated with a lower metabolic demand than concentric actions (Dudley et al., 1991a; Ryschon et al., 1997) as well as lower cardiovascular responses (Hortobagyi et al, 2001; MacDougall et al., 1991; Trinkwon et al., 2001). While not directly measured during the current resistance training investigation, metabolic and cardiovascular stress was likely lower with the LE protocol, as suggested by unaltered cortisol levels in LE. Lower metabolic and cardiovascular stress, combined with increased dynamic strength may be especially relevant to the elderly, people with disabilities, chronic disease states or undergoing rehabilitation from accident or injury, where increased strength could assist with independent function.

A few additional considerations regarding both the LC and LE training protocols involve the lower relative training intensity and the total time per session. The use of lower loads during training may be a safety consideration, especially with some populations.

Each repetition required 10 s to complete, which is longer than "usual". As a result, fewer sets were required to achieve significant and substantial adaptations, with the total training time per session reduced. Finally, it must be considered that the results of the current investigations were found with young healthy adult females and may not be replicated with other populations.

REFERENCES

Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Halkjær-Kristensen J & Dyhre-Poulsen P (2000) Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. J Appl Physiol 89(6): 2249-2257.

Adams GR & Haddad F (1996) The relationships among IGF-1, DNA content, and protein accumulation during skeletal muscle hypertrophy. J Appl Physiol 81(6): 2509-2516.

Adams GR, Hather BM, Baldwin KM & Dudley GA (1993) Skeletal muscle myosin heavy chain composition and resistance training. J Appl Physiol 74(2): 911-915.

Amiridis IG, Martin A, Morlon B, Martin L, Cometti G, Pousson M & van Hoecke J (1996) Co-activation and tension-regulating phenomena during isokinetic knee extension in sedentary and highly skilled humans. Eur J Appl Physiol (73): 149-156.

Anderson T & Kearney JT (1982) Effects of three resistance training programs on muscular strength and absolute and relative endurance. Res Q Exercise Sport: 53(1), 1-7.

Asmusen E (1953) Positive and negative muscular work. Acta Physiol Scand 28: 364-382.

Aura O & Komi PV (1986) Mechanical efficiency of pure positive and pure negative work with special reference to the work efficiency. Int J Sports Med 7: 44-49.

Babault N, Pousson M, Michaut A, Ballay Y & Van Hoecke J (2002) EMG activity and voluntary activation during knee-extensor concentric torque generation. Eur J Appl Physiol (86): 541-547.

Baker D (1995) The effect of a wave-like periodized strength training cycle on maximal strength in men. Strength Cond Coach 3(3): 11-16.

Baldi JC & Reiser PJ (1995) Intermediate filament proteins increase during chronic stimulation of skeletal muscle. J Muscle Res Cell Motility 16: 587-594.

Behm DG & Sale DG (1996) Influence of velocity on agonist and antagonist activation in concentric dorsiflexion muscle actions. Can J Appl Phys 21(5): 402-416.

Bell GJ, Syrotuik D, Martin TP & Burnham R (2000) Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. Eur J Appl Physiol (81): 418-427.

Bell G, Syrotuik D, Socha T, MacLean I & Quinney HA (1997). Effect of strength training and concurrent strength and endurance training on strength, testosterone and cortisol. J Strength Cond Res 11: 57-64.

Bell GJ & Wenger HA (1992) Physiological adaptations to velocity-controlled resistance training. Sports Med 13(4): 234-244.

Ben-Sira D, Ayalon A & Tavi M (1995) The effect of different types of strength training on concentric strength in women. J Strength Cond Res 9(3): 143-148.

Bigland B & Lippold OCJ (1954) The relation between force, velocity and integrated electrical activity in human muscles. J Physiol (Lond) 123: 214-224.

Bigland-Ritchie B (1981) EMG/force relations and fatigue of human voluntary contractions. Exerc Sport Sci Res 9: 77-117.

Bigland-Ritchie B & Woods JJ (1974) Integrated EMG and oxygen uptake during dynamic contractions of human muscles. J Appl Physiol 36: 475-479.

Bigland-Ritchie B & Woods JJ (1976) Integrated electromyogram and oxygen uptake during positive and negative work. J Physiol (Lond) 260: 267-277.

Blomstrand E & Ekblom B (1982) The needle biopsy technique for fibre type determination in human skeletal muscle – a methodological study. Acta Physiol Scand 116: 437-442.

Bornemann A & Schmalbruch H (1992) Desmin and vimentin in regenerating muscles. Muscle Nerve 15: 14-20.

Brandenburg JP & Docherty D (2002) The effects of accentuated eccentric loading on strength, muscle hypertrophy, and neural adaptations in trained individuals. J Strength Cond Res 16(1): 25–32.

Carey Smith R & Rutherford OM (1995) The role of metabolites in strength training I. A comparison of eccentric and concentric contractions. Eur J Appl Physiol 71: 332-336.

Carroll TJ, Abernethy PJ, Logan PA, Barber M & McEniery (1998) Resistance training frequency: strength and myosin heavy chain responses to two and three bouts per week. Eur J Appl Physiol 78: 270-275.

Chilibeck PD, Syrotuik DG & Bell GJ (1999) The effect of strength training on estimates of mitochondrial density and distribution throughout muscle fibres. Eur J Appl Physiol 80: 604-609.

Clarkson PM, Nosaka K & Braun B (1992) Muscle function after exercise-induced muscle damage and rapid adaptation. Med Sci Sports Exerc 24(5): 512-520.

Colliander EB & Tesch PA (1990). Effects of eccentric and concentric muscle actions in resistance training. Acta Physiol Scand 140: 31-39.

Cronin JB, McNair PJ & Marshall RN (2001) Magnitude and decay of stretch-induced enhancement of power output. Eur J Appl Physiol 84(6): 575-581.

Cureton KJ, Collins MA, Hill DW & McElhannon FM Jr. (1988) Muscle hypertrophy in men and women. Med Sci Sports Exerc 20(4): 338-344.

Dalton NJ & Wallace JE (1996) Strength testing protocols for college-age women. Strength and Cond J 18(2): 1996.

Dalton PA & Stokes MJ (1991) Acoustic myography reflects force changes during dynamic concentric and eccentric contractions of the human biceps brachii muscle. Eur J Appl Physiol Occup Physiol 63: 412-416.

Donne B & Luckwill RG (1996) Co-activation of quadriceps and hamstring muscles during concentric and eccentric isokinetic exercise. Isokinetics Exerc Sci 6(1): 21-26.

Dons B, Bollerup K, Bonde-Petersen F & Hancke S (1979). The effect of weight-lifting exercise related to muscle fiber cross-sectional area in humans. Eur J Appl Physiol Occup Physiol 40: 95-106.

Dudley GA, Tesch PA, Harris RT, Golden CL & Buchanan P (1991a) Influence of eccentric actions on the metabolic cost of resistance exercise. Aviat Space Environ Med 62: 678-682.

Dudley GA, Tesch PA, Miller BJ & Buchanan P (1991b) Importance of eccentric actions in performance adaptations to resistance training. Aviat Space Environ Med 62: 543-550.

Dunn SE, Burns JL & Michel RN (1999) Calcineurin is required for skeletal muscle hypertrophy? J Biol Chem 274(31): 21908-21912.

Ebbeling CB & Clarkson PM (1989) Exercise-induced muscle damage and adaptation. Sports Med 7: 207-234.

Enoka RM (1996) Eccentric contractions require unique activation strategies by the nervous system. J Appl Physiol 81(6): 2339-2345.

Enoka RM (1988) Muscle strength and its development. Sports Med 6: 146-168.

Evans WJ, Phinney SD & Young VR (1982) Suction applied to a muscle biopsy maximizes sample size. Med Sci Sports Exerc 14(1): 101-102.

Fang Y, Siemionow V, Sahgal V, Xiong F & Yue GH (2001) Greater movement-related cortical potential during human eccentric versus concentric muscle contractions. J Neurophysiol 86: 1764-1772.

Farthing JP & Chilibeck PD (2003) The effects of eccentric and concentric training at different velocities on muscle hypertrophy. Eur J Appl Physiol 89: 578-586.

Faulkner JA, Brooks SV & Opiteck JA (1993) Injury of skeletal muscle fibres during contractions: conditions of occurrence and prevention. Phys Ther 73(12): 911-921.

Fiatarone Singh MA, Ding W, Manfredi TJ, Solares GS, O'Neill E F, Clements KM, Ryan ND, Kehayias JJ, Fielding RA & Evans WJ (1999) Insulin –like growth factor I in skeletal muscle after weight-lifting exercise in frail elders. Am J Physiol 277(1): E135-E143.

Fridén J, Kjörell U & Thornell L-E (1984) Delayed muscle soreness and cytoskeletal alterations: An immunocytological study in man. Int J Sports Med (5): 15-18.

Fridén J, Seger J & Ekblom B (1988) Sublethal muscle fibre injuries after high-tension anaerobic exercise. Eur J Appl Physiol 57: 360-368.

Fridén J, Sjöström M & Ekblom B (1983) Myofibrillar damage following intense eccentric exercise in man. Int J Sports Med 4: 170-176.

Franzini-Armstrong C & Fischman W (1994) Myology: basic and clinical. AG Engel & C Franzini-Armstrong (Eds.). New York: McGraw Hill.

Frontera WR, Meredith CN, O'Reilly KP, Knuttgen HG & Evans WJ (1988) Strength conditioning in older men: skeletal muscle hypertrophy and improved function. J Appl Physiol 64(3): 1038-1044.

Gibala MJ, MacDougall JD, Tarnopolsky MA, Stauber WT & Elorriaga A (1995) Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise. J Appl Physiol 78(2): 702-708.

Gibala MJ, Interisano SA, Tarnopolsky MA, Roy BD, MacDonald JR, Yarasheski KE & MacDougall JD (2000) Myofibrillar disruption following acute concentric and eccentric resistance exercise in strength-trained men. Can J Physiol Pharmacol 78: 656-661.

Gillies EM & Docherty D (1999) Effects of different times of concentric muscle actions on strength, hypertrophy, specific tension, and training volume in resistance trained women. Med Sci Sports Exerc 31(5): S114.

Gillies EM, Zehr EP, Bell GJ, Haridas C & Sugajima Y (2000) Muscle activation during lengthening and shortening plantar flexion of varying duration with a submaximal load. Can J Appl Physiol 25(5): 374.

Goldberg AL, Etinger JD, Goldspink DF & Jablecki C (1975) Mechanism of work-induced hypertrophy of skeletal muscle. Med Sci Sports Exerc 7(4): 248-261.

Golden CL & Dudley DA (1992) Strength after bouts of eccentric and concentric actions. Med Sci Sports Exerc 24(8): 926-933.

Gulch RW (1994) Force-velocity relations in human skeletal muscle. Int J Sports Med 15: S2-S10.

Gulling LK, Lephart SM, Stone DA, Irrgang JJ & Pincivero DM (1996) The effects of patellar bracing on quadriceps EMG activity during isokinetic exercise. Isokinetics Exerc Sci 6(2): 133-138.

Hadley ME (1996) Adrenal steroid hormones. Endocrinology (pp. 338-369). Toronto: Prentice-Hall Canada.

Häkkinen K & Komi PV (1981) Effect of different combined concentric and eccentric muscle work regimens on maximal strength development. J Human Movement Studies 7: 33-44.

Häkkinen K, Komi PV & Alén M (1985). Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. Acta Physiol Scand 125: 587-600.

Hawke TJ & Garry DJ (2001) Myogenic satellite cells: physiology to molecular biology. J Appl Physiol 91: 534-551.

Hayward R, Dennehy CA, Rodearmel SJ & Schneider CM (1998) Serum creatine kinase, CK-MB, and perceived soreness following eccentric exercise in oral contraceptive users. Sports Med Training Rehab 8(2): 193-207.

Hather BM, Tesch PA, Buchanan P & Dudley GA (1991) Influence of eccentric actions on skeletal muscle adaptations to resistance training. Acta Physiol Scand 143: 177-185.

Hernandez JM, Fedele MJ & Farrell PA (2000) Time course evaluation of protein synthesis and glucose uptake after acute resistance exercise in rats. J Appl Physiol 88: 1142-1149.

Hickson JF Jr., Wolinsky I, Rodriguez GP, Pivarnik JM, Kent MC & Shier N (1986) Failure of weight training to affect urinary indices of proteins metabolism in men. Med Sci Sports Exerc 18(5): 563-567.

Hoeger WWK, Hopkins DR, Barette SL & Hale DF (1990) Relationship between repetitions and selected percentages of one repetition maximum: A comparison between untrained and trained males and females. J Appl Sport Sci Res 4(2): 47-54.

Hortobágyi T, Barrier J, Beard D, Braspennincx J, Koens P, Devita P, Dempsey L & Lambert J (1996) Greater initial adaptations to submaximal muscle lengthening than maximal shortening. J Appl Physiol 81(4): 1677-1682.

Hortobágyi T, Devita P, Money J & Barrier J (2001) Effects of standard and eccentric overload strength training in young women. Med Sci Sports Exerc 33(7): 1206-1212.

Hortobágyi T, Houmard J, Fraser D, Dudek R, Lambert J & Tracy J (1998) Normal forces and myofibrillar disruption after repeated eccentric exercise. J Appl Physiol 84(2): 492-498.

Housh DJ, Housh TJ, Weir JP, Weir LL, Evetovich TK & Donlin PE (1998a) Effects of unilateral concentric-only dynamic constant external resistance training on quadriceps femoris cross-sectional area. J Strength Cond Res 12(3): 185-191.

Housh DJ, Housh TJ, Weir JP, Weir LL, Evetovich TK & Donlin PE (1998b) Effects of unilateral eccentric-only dynamic constant external resistance training on quadriceps femoris cross-sectional area. J Strength Cond Res 12(3): 192-198.

Howell JN, Fuglevand AJ, Walsh ML & Bigland-Ritchie B (1995) Motor unit activity during isometric and concentric-eccentric contractions of the human first dorsal interosseus muscle. J Neurophysiol 72(2): 901-904.

Huszar G, Golenwsky G, Maiocco J & Davis E (1983) Urinary 3-methylhistidine excretion in man: the role of protein-bound and soluble 3-methylhistidine. Br J Nutr 49: 287-294.

Johnson BL (1972) Eccentric vs concentric muscle training for strength development. Med Sci Sports Exerc 4(2): 111-115.

Johnson BL, Adamczyk JW, Tenøe KO & Strømme SB (1976) Comparison of concentric and eccentric muscle training. Med Sci Sports Exerc 8(1): 35-38.

Jones DA, Rutherford OM & Parker DF (1989) Physiological changes in skeletal muscle as a result of strength training. Q J Exp Physiol 74: 233-256.

Jürimäe L, Abernethy PJ, Blake K & McEniery MT (1996) Changes in the myosin heavy chain isoform profile of the triceps brachii muscle following 12 weeks of resistance training. Eur J Appl Physiol 74: 287-292.

Kadi F, Ahlgren C, Waling K, Sundelin G & Thornell L-E (2000) The effects of different training programs on the trapezius muscle of women with work-related neck and shoulder myalgia. Acta Neuropathol 100: 253-258.

Kadi F & Thornell L-E (2000) Concomitant increases in myonuclear and satellite cell content in female trapezius muscle following strength training. Histochem Cell Biol 113: 99-103.

Kadi F & Thornell L-E (1999) Training affects myosin heavy chain phenotype in the trapezius muscle of women. Histochem Cell Biol 112: 73-78.

Katz B (1939) The relation between force and speed in muscular contraction. J Physiol (Lond) (96): 45-64.

Kellis E & Baltzopoulos V (1998) Muscle activation differences between eccentric and concentric isokinetic exercise. Med Sci Sports Exerc 30(11):1616-1623.

Komi PV, Linnamo, VA, Ventoinen PS & Sillanpaa M (2000) Force and EMG power spectrum during eccentric and concentric actions. Med Sci Sports Exerc 32(10): 1757-1762.

Komulainen J, Kalliokoski R, Koskinen SOA, Drost MR, Kuipers H & Hesselink MKC (2000) Controlled lengthening or shortening contraction induced damage is followed by fiber hypertrophy in rat skeletal muscle. Int J Sports Med 21: 107-112.

Komulainen J, Koskinen SOA, Kalliokoski R, Takala TES & Vihko V (1999) Gender differences in skeletal muscle fibre damage after eccentrically biased downhill running in rats. Acta Phsyiol Scand 165: 57-63.

Lemon PWR (1994) Are dietary protein needs affected by regular exercise? Sports Nutrition Foundation 2(3): 1994.

Lieber RL & Fridén J (1993) Muscle damage is not a function of muscle force but active strain. J Appl Physiol 74(2): 520-526.

Lieber RL, Thornell L & Fridén J (1996) Muscle cytoskeletal disruption occurs within the first 15 min of cyclic eccentric contraction. J Appl Physiol 80(1): 278-284.

Linnamo V, Strojnik V & Komi PV (2002) EMG power spectrum and features of the superimposed M-wave during voluntary eccentric and concentric actions at different activation levels. Eur J Appl Physiol 86: 534-540.

Lynch GS, Rafael JA, Chamberlain JS & Faulkner JA (2000) Contraction-induced injury to single permeabilized muscle fibers from mdx, transgenic mdx, and control mice. Am J Physiol Cell Physiol 279: C1290-C1294.

MacDougall JD, McKelvie RS, Moroz DE, Sale DG, McCartney N & Buick F (1992) Factors affecting blood pressure during heavy weight lifting and static contractions. J Appl Physiol 73(4): 1590-1597.

Macpherson PCD, Schork MA & Faulkner JA (1996) Contraction-induced injury to single fiber segments from fast and slow muscles of rats by single stretches. Am J Physiol 271 (Cell Physiol 40): C1438-C1446.

McCall GE, Byrnes WC, Dickinson AL & Fleck SJ (1998) Sample size required for the accurate determination of fiber area and capillary of human skeletal muscle. Can J Appl Physiol 23(6): 594-599.

McHugh MP, Tyler TF, Greenberg SC & Gleim GW (2002) Differences in activation patterns between eccentric and concentric quadriceps contractions. J Sports Sci (20): 83-91.

Mikesky AE, Matthews W, Giddings CJ & Gonyea WJ (1989) Muscle enlargement and exercise performance in the cat. J Appl Sport Sci Res 3(4): 85-92.

Moritani T (1992) Time course of adaptations during strength and power training. In PV Komi (Ed.) Strength and power in sport (pp. 266-278). London: Blackwell Scientific Publications.

Morrissey MC, Harmon EA, Frykman PN & Han KH (1998) Early phase differential effects of slow and fast barbell squat training. Am J Sports Med 26(2): 221-230.

Nakazawa K, Kawakami Y, Fukunaga T, Yano H & Miyashita M (1993) differences in activation patterns in elbow flexor muscles during isometric, concentric and eccentric contractions. Eur J Appl Physiol 66(3): 214-220.

Nardone A, Romano C & Schieppati M (1989) Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscles. J Physiol (Lond) 409: 451-471.

Narici MV, Hoppeler H, Kayser B, Landoni L, Claassen H, Gavardi C, Conti M & Cerretelli P (1996) Human quadriceps cross-sectional area, torque and neural activation during 6 months strength training. Acta Physiol Scand 157: 175-186.

Narici MV, Roi GS, Landoni L, Minetti AE & Cerretelli P (1989) Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. Eur J Appl Physiol 59: 310-319.

Newham DJ, Jones DA & Clarkson PM (1987) Repeated high-force eccentric exercise: effects on muscle pain and damage. J Appl Physiol 63(4): 1381-1386.

Newham DJ, Mills KR, Quigley BM & Edwards RHT (1983) Pain and fatigue after concentric and eccentric muscle contractions. Clin Sci 64: 55-62.

Nissen S, Sharp R, Ray M, Rathmacher JA, Rice D, Fuller Jr. JC, Connelly AS & Abumrad N (1996) Effect of leucine metabolite β-hydroxy-β-methylbutyrate on muscle metabolism during resistance-exercise training. J Appl Physiol 81(5): 2095-2104.

Nosaka K & Clarkson PM (1995) Muscle damage following repeated bouts of high force eccentric exercise. Med Sci Sports Exerc 27(9): 1263-1269.

O'Hagan FT, Sale DG, MacDougall JD & Garner SH (1995) Responses to resistance training in young women and men. Int J Sports Med 16: 314-321.

Oskowski KJ, Wilson GJ, Weatherby R, Murphy PW & Lyttle AD (1995) The effect of weight training volume on hormonal output and muscular size and function. J Strength Cond Res 11(3): 148-154.

Palmieri GA (1987) Weight training and repetition speed. J Appl Sport Sci Res 1: 36-38.

Paul GL, DeLany JP, Snook JT, Seifert JG & Kirby TE (1989) Serum and urinary markers of skeletal muscle tissue damage after weight lifting exercise. Eur J Appl Physiol 58: 786-790.

Patel TJ & Lieber RL (1997) Force transmission in skeletal muscle: from actomyosin to external tendons. Exerc Sport Sci Rev: 321-363.

Pette D & Staron RS (2000) Myosin isoforms, muscle fiber types, and transitions. Microsc Res Tech 50: 500-509.

Perrine JJ & Edgerton VR (1978) Muscle force-velocity and power-velocity relationships under isokinetic loading. Med Sci Sports 10:159-166.

Petrof BJ, Shrager JB, Stedman HH, Kelly AM & Sweeney HL (1993) Dystrophin protects the sarcolemma from stresses developed during muscle contraction. Proc Natl Acad Sci 90: 3710-3714.

Prou E, Guevel A, Benezet P & Marini JF (1999) Exercise-induced muscle damage: Absence of adaptive effect after a single session of eccentric isokinetic heavy resistance exercise. J Sports Med Phys Fitness 39: 226-232.

Putman CT, Düsterhöft S & Pette D (2000) Satellite cell proliferation in low frequency-stimulated fast muscle of hypothyroid rat. Am J Physiol Cell Physiol 279: C682-C690.

Roth SM, Martel GF, Ivey FM, Lemmer JT, Metter EJ, Hurley BF & Rodgers MA (2000) High-volume, heavy-resistance strength training and muscle damage in young and older women. J Appl Physiol 88(3): 1112-1118.

Roth S M, Martel G F, Ivey F M, Lemmer J T, Tracy B L, Hurlbut D E, Metter E J, Hurley B F & Rodgers M A (1999) Ultrastructrual muscle damage in young vs. older men after high-volume, heavy-resistance strength training. J Appl Physiol 86(6): 1883-1840.

Roth S M, Martel G F, Ivey F M, Lemmer J T, Tracy B L, Metter E J, Hurley B F & Rodgers M A (2001) Skeletal muscle satellite cell characteristics in young and older men and women after heavy resistance strength training. J Gerontol Biol Sci 56A(6): B240-B247.

Ryschon TW, Fowler MD, Wysong RE, Anthony AR. & Balaban RS (1997) Efficiency of human skeletal muscle in vivo: comparison of isometric, concentric, and eccentric muscle action. J Appl Physiol 83(3): 867-874.

Sale DG (1988) Neural adaptation to resistance training. Med Sci Sports Exer 20(5): S135-S145.

Schachat F, Williams RS & Schnurr CA (1988) Coordinate changes in fast thin filament and Z-line protein expression in the early response to chronic stimulation. J Biol Chem 263(28): 13975-13978.

Schott J (1995) The role of metabolites in strength training: short versus long isometric contractions. Eur J Appl Physiol 71(4): 337-341.

Seliger V, Dolejš L, Karas V & Pachlopníková I (1968) Adaptation of trained athletes' energy expenditure to repeated concentric and eccentric muscle contractions. Int Z angew Physiol Einschl Arbeitsphysiol 26: 227-234.

Sharman MJ, Newton RU, Triplett-McBride T, McGuigan MRM, McBride JM, Häkkinen A, Häkkinen K & Kraemer WJ (2001) Changes in myosin heavy chain composition with heavy resistance training in 60- to 75-year-old men and women. Eur J Appl Physiol 84: 127-132.

Sorichter S, Mair J, Koller A, Gebert W, Rama D, Calzolari C, Artner-Dworzak E & Puschendorf B (1997a) Skeletal tropinin I as a marker of exercise-induced muscle damage. J Appl Physiol 83(4): 1076-1082.

Sorichter S, Mair J, Koller A, Secnik P, Parrak V, Haid C, Müller E & Puschendorf B (1997b) Muscular adaptation and strength during the early phase of eccentric training: influence of the training frequency. Med Sci Sports Exerc 29(12): 1646-1652.

Staron RS, Hikida RS, Murray TF, Nelson MM, Johnson P & Hagerman F (1992) Assessment of skeletal muscle damage in successive biopsies from strength-trained and untrained men and women. Eur J Appl Physiol 65: 258-264.

Staron RS, Karapondo DL, Kraemer WJ, Fry AC, Gordon SE, Falkel JE, Hagerman FC & Hikida RS (1994). Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. J Appl Physiol 76(3): 1247-1255.

Staron RS, Leonardi M J, Karapondo DL, Malicky ES, Falkel JE, Hagerman FC & Hikida RS (1991) Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. J Appl Physiol 70(2): 631-640.

Staron RS, Malicky ES, Leonardi MJ, Falkel JE, Hagerman FC & Dudley GA (1989) Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. Eur J Appl Physiol (60): 71-79.

Stauber WT (1989) Eccentric action of Muscles: Physiology, injury, and adaptation. Exerc Sport Sci Rev 17: 157-185.

Takekura H & Yoshioka T (1990) Ultrastructural and metabolic characteristics of single muscle fibres belonging to the same type in various muscles in rats. J Muscle Res Cell Motility (11): 98-104.

Tax AAM, Denier van der Gon JJ, Gielan CCAM & van den Tempel CMM (1989) Differences in the activation of m. Biceps brachii in the control of slow isotonic movements and isometric contractions. Exp Brain Res 76: 55-63.

Termin A, Staron RS & Pette D (1989) Changes in myosin heavy chain isoforms during CLFS of rat fast hindlimb muscles – a single fiber study. Eur J Biochem 186: 749-754.

Theeuwen M, Gielen DDAM & Miller LE (1994) The relative activation of muscles during isometric contractions and low-velocity movements against a load. Exp Brain Res 101: 493-505.

Tiidus PA (2000) Estrogen and gender effects on muscle damage, inflammation, and oxidative stress. Can. J Appl Physiol 25(4): 274-287.

Toumi H, Thiery C, Maitre S, Martin A, Vanneuville G & Poumarat G (2001) Training effects of the amortization phase on the vertical jump. Int J Sports Med (22): 605-610.

Trinkwon HA, Woltz BD, Overend TJ & Vandervoort AA (2001) Cardiovascular responses to maximal isokinetic concentric and eccentric knee extension exercise in young adults. Can J Appl Physiol 26(5): 519.

Urhausen A, Gabriel H & Kindermann W (1995) Blood hormones as markers of training stress and overtraining. Sports Med. 20(4): 251-276.

Villani R (1987) The effects of progressive resistance training on skeletal muscle protein degradation in man. Australian J Sci Med Sport 19(3): 14-17.

Viru A, Litvinova L, Viru M & Smirnova T (1994) Glucocorticoids in metabolic control during exercise: alanine metabolism. J Appl Physiol 76: 801-805.

Viru A & Seli N (1992) 3-methylhistidine excretion in training for improved power and strength. Sports Med Training Rehab 3: 183-193.

Waterman-Storer C (1991) The cytoskeleton of skeletal muscle: is it affected by exercise? A brief review. Med Sci Sports Exerc 23(11): 1240-1249.

Webber S & Kriellers D (1997) Neuromuscular factors contributing to *in vivo* eccentric moment generation. J Appl Physiol 78: 555-559.

Weiss LW, Clark FC & Houmard DG (1988) Effects of heavy-resistance triceps surae muscle training on strength and muscularity of men and women. Phys Ther 68(2): 208-213.

Westing SH, Cresswell AG & Thorstensson A (1991) Muscle activation during maximal voluntary eccentric and concentric knee extension. Eur J Appl Physiol 1991 62: 104-108.

Westing S, Seger J & Thorstensson A (1990) Effects of electrical stimulation on eccentric and concentric torque-velocity relationships during knee extension in man. Acta Physiol Scand 140: 17-22.

Williamson DL, Gallagher PM, Carroll CC, Raue U & Trappe SW (2001) Reduction in hybrid single muscle fiber proportions with resistance training in humans. J Appl Physiol 91: 1955-1961.

Wilson G J (1994) Strength and power in sport. In J Bloomfield, TR Ackland & BC Elliot (Eds.) Applied Anatomy and Biomechanics in Sport, (pp.110-208). Melbourne: Blackwell Scientific Publications.

Wilson GJ, Elliott BC & Wood GA (1991) The effect on performance of imposing a delay during a stretch-shorten cycle movement. Med Sci Sports Exerc 23(3): 364-370.

Wilson GJ, Murphy AJ & Giorgi A (1996) Weight and plyometric training: Effects on eccentric and concentric force production. Can J Appl Physiol (21(4): 301-315.

Young WB & Bilby GE (1993) The effect of voluntary effort to influence speed of contraction on strength, muscular power, and hypertrophy development. J Strength Cond Res 7: 172-178.

Young V & Munro HN (1978) N^{τ} - Methylhistidine (3-methylhistidine) and muscle protein turnover: an overview. Fed Proc 37: 2291-2300.

Yuhasz, MS (1966) Physical Fitness and Sports Appraisal Laboratory Manual, London, Canada: The University of Western Ontario.

APPENDIX A MUSCLE ACTIVATION DURING ECCENTRIC AND CONCENTRIC MUSCLE ACTIONS OF VARYING DURATION WITH A SUBMAXIMAL LOAD[1]

Introduction

A difference in neural activity occurs between concentric muscle actions, where tension is generated by a muscle as it shortens, and eccentric muscle actions, which generate tension in the muscle while the muscle lengthens. This has led to the suggestion that there is a different neural control strategy for muscle actions, that is dependent on whether the muscle is generating tension by shortening or lengthening (Enoka, 1996).

There have been a number of different approaches employed to examine neural activity during concentric and eccentric muscle actions. A number of investigators have chosen to compare muscle activation, assessed by surface electromyogropahy (EMG), during dynamic anisometric concentric and eccentric muscle actions using the same external load and the same movement velocity. Results from these studies indicate that lower EMG levels occur during eccentric actions. This has been observed with the load varying from zero to near maximal for individual muscles of synergist muscle groups such as the elbow flexors (Dalton & Stokes, 1991; Fang et al., 2001; Nakazawa et al., 1993; Nakazawa et al., 1997) and leg extensors (Aura & Komi, 1986). However, in all of these investigations, neural activity during concentric and eccentric muscle actions were compared at one velocity only.

---

[1] This investigation was completed with the assistance of Dr. E. P. Zehr.

Another approach to examining neural activity of concentric and eccentric actions has been to use an isokinetic action along with maximal levels of force. As with anisometric investigations using submaximal loads, neural activity during isokinetic concentric actions also appears to be associated with greater EMG than that of eccentric actions at the same velocity (Donne & Luckwill, 1996; Gulling et al., 1996; Kellis & Baltzopoulos, 1998; Komi et al., 2000; Westing et al., 1991) despite the greater levels of torque generated during eccentric muscle actions. The influence of velocity on neural activity has also been investigated, mainly with the leg extensors muscle group, during maximal isokinetic actions. Information regarding the effects of velocity on muscle activation during maximal concentric actions is conflicting. As movement velocity increases, EMG has been shown to increase (Westing et al., 1991), stay the same (Kellis & Baltzopoulos, 1998; Komi et al., 2000), or to demonstrate an inverse relationship (Behm & Sale, 1996). EMG during maximal eccentric actions appears to remain the same irrespective of velocity (Kellis & Baltzopoulos, 1998; Komi et al., 2000; Westing et al., 1991).

There have been a few investigations that have looked at the influence of velocity on muscle activation during dynamic anisometric actions with a submaximal external load. These investigations have all focused on muscles of the triceps surae during plantar flexion at velocities of 1.05 rad · s^{-1} and slower. Bigland and Lippold (1954) investigated the relationship between muscle activity and velocity during concentric and eccentric muscle actions with a load of 3.75 kg. They found that activity of the gastrocnemius muscle (no distinction was made between the different heads) increased with concentric velocity but velocity had no effect on eccentric muscle activity. Statistical significance was not reported. Romano and Schieppati (1987), in an investigation into soleus H-reflex response during concentric and eccentric muscle actions, also reported on the behavior of the soleus during concentric and eccentric plantar flexion. The external load was fixed at 100 N, corresponding to approximately 15-20% of maximal voluntary contraction (MVC). They reported that EMG increased with concentric velocity but decreased with eccentric velocity. Furthermore, velocity influenced EMG to a greater extent during concentric than eccentric actions, and as well, EMG increased throughout the movement during concentric actions while the opposite pattern was found during eccentric actions. The data analysis was quite qualitative, with statistical significance not reported.

The triceps surae muscle group (comprised of the two heads of the gastrocnemius and the soleus muscles) are the primary muscles involved in plantar flexion. These muscles differ in muscle fiber type composition (Alway et al., 1989; Johnson et al., 1973) and architectural characteristics, even between the two heads of the gastrocnemius (Kawakami et al., 1998; Nichols, 1994). Thus the triceps surae provides an interesting opportunity to examine the relative activation of synergist muscles during concentric and eccentric muscle actions.

The objective of this investigation was to evaluate the effects of velocity of movement on EMG during non-fatiguing dynamic concentric and eccentric plantar flexion performed with a submaximal load. Expansion of previous investigations was accomplished by examining the behavior of all muscles of the triceps surae muscle group as well as the antagonist tibialis anterior muscle over a range of velocities. In addition, a greater load and a more quantitative approach than previous investigations were used. It was hypothesized that velocity would influence EMG activity during concentric, but not during eccentric actions and that concentric activation would be greater than eccentric activation at the same velocity. Based upon other documented evidence of possible selective activation of "fast" motor units during eccentric actions (Nardone et al., 1989), it was hypothesized that the gastrocnemius muscles would be more active than soleus during eccentric actions. A preliminary account of this work has appeared as an abstract (Gillies et al., 2000).

Methods

Subjects

Ten healthy physically active subjects free of any known neuromuscular disorder volunteered to participate in this study. Subjects (5 women and 5 men) had an average (SD) age of 31.3 (7.2) yr, height of 170.0 (10.6) cm and body mass of 74.5 (14.8) kg. Written informed consent was obtained from all subjects in accordance with the requirements of the Faculty of Physical Education and Recreation Research Ethics Committee at the University of Alberta.

Experimental Setup

Subjects were seated in a straight-back chair mounted on an elevated platform with their right foot placed in the footplate of a custom designed apparatus that allowed isometric and dynamic plantarflexion and dorsiflexion movements against an inertial load. The foot was firmly strapped to the metal plate so that during all movements, full contact between the foot and plate was maintained. The height and depth of the footplate were adjusted so that the lateral malleolus was aligned with the axis of rotation of the attachment of the footplate to the frame of the apparatus. The left foot rested on the platform on which the chair and apparatus were attached. The position of the chair on the platform was adjustable so that the knee joint was flexed at an angle of 1.22 rad (0 rad full extension) and the ankle joint at 1.57 rad (neutral position with the foot and tibia forming a right angle, at rest, the start and end points of the concentric and eccentric actions, respectively, and during the isometric maximal efforts). The dynamic muscle actions were performed over a range of motion (ROM) of 0.52 rad of plantarflexion from the neutral position (range varied from 0.37 to 0.68 rad) (Fig.1). A load was attached through the rear of the footplate through a stainless steel cable and a pulley fixed to the frame of the apparatus. Concentric actions were performed by lifting the load, and eccentric actions by lowering the load. The free end of the cable could be fixed in place for isometric actions. A strain gauge force transducer was inserted in series with the cable between the footplate and the pulley. Positional information was supplied by a potentiometer attached to the axis of rotation of the footplate. Both position and force signals could be displayed on an oscilloscope mounted to the frame in front of the subjects.

Experimental Procedures

Two sessions were required of each subject. A familiarization session occurred 5-7 days prior to the experimental session. During this first session, each subject performed several isometric plantarflexion maximal voluntary contractions (MVC). The MVC task consisted of an isometric sustained effort for 5 s to ensure that peak force was attained. Subjects were verbally encouraged to increase their force output and they were able to monitor their force level on the oscilloscope display. The upper body was not restrained during the MVC efforts but the subjects held their arms in a crossed position with their hands on their shoulders.

Following the MVC, subjects practiced concentric and eccentric plantarflexion movements, performed in isolation, at attempted average velocities of 0.18, 0.35, 0.52, 0.87, 1.31, and 2.09 rad $\cdot$ s^{-1} using a load of approximately 30% of MVC. Subjects were asked to control the raising and lowering of the load by attempting to maintain a constant velocity throughout the movement and by matching a velocity template on the oscilloscope, set to display angular displacement. Subjects were allowed to practice each condition (total of 12 conditions, 6 velocity x 2 muscle actions) until they felt that they were able to match the template. This required approximately 5-10 repetitions. For the eccentric conditions, the load was held isometrically for several seconds before it was lowered.

The experimental session was similar to the familiarization session except that a dorsiflexion MVC was performed in addition to the plantarflexion MVC. The different conditions were presented in a random order, with 1 min rest between trials. Subjects were allowed to perform as many trials as necessary to obtain five acceptable trials. After each trial, the movement pattern and EMG signals were displayed on a computer monitor. A trial was acceptable if the movement pattern appeared to be of even velocity and matched other trials for that subject. As well, the trials that contained high tibialis anterior (TA) activation were also discarded. When this did occur, it occurred during eccentric actions.

For most conditions, subjects were able to perform 5 consecutive similar trials, although one or two additional trials were required more often at the faster velocities. Data for a given subject were averaged across the five trials.

Data Collection

Surface electromyographic (EMG) signals, along with force and joint angle recordings were simultaneously collected, processed and stored for later analysis (see sample data from a representative subject plotted in Fig. A-1). Each instrument was calibrated immediately before data collection. EMG was collected from the agonist triceps surae muscles; soleus (Sol) and the lateral (LG) and medial (MG) gastrocnemius, and antagonist TA of the right leg. The skin was lightly abraded and cleaned with an isopropyl alcohol swab prior to electrode attachment. Bipolar Ag-AgCl surface electrodes (Kendall-LTP, Chicopee, MA) (2 cm inter-electrode distance) were attached over the belly of each muscle along the predicted path of the fibres and a ground electrode was placed over the patella. The raw EMG signals were amplified (x 500-5,000) and band-pass filtered at 30-300 Hz (P511 Grass Instruments, AstroMed, Inc., West Warwich, RI). Data from each EMG channel, potentiometer and strain gauge were sampled at a rate of 1000 Hz with a 12-bit A/D converter connected to a computer system running custom-written (Dr. Romeo Chua, University of British Columbia) LabVIEW (National Instruments, Austin, TX) virtual instruments.

Data Analysis

All data collected during the experiments were recorded and stored until analyzed with Matlab (The MathWorks Inc., Natick, MA) using custom-written software (Mr. A. Ley). EMG of the agonist muscles was normalized to the respective muscle during MVC plantarflexion and antagonist EMG was normalized to MVC during dorsiflexion. All dependent variables were related to the ROM. These included the average EMG amplitude (aEMG, calculated as the normalized EMG, divided by the movement time), peak EMG (pEMG), time of pEMG occurrence, normalized to total movement time, and peak and average velocity. The ROM was further subdivided into 10 bins with each bin equal to 1/10 the total movement time. Bin data were examined for the variables of aEMG, velocity , acceleration and force.

Muscle activity on-set (concentric) and off-set (eccentric) was determined as the time from the start of movement at which muscle activity increased over baseline EMG levels (on-set), or the time from the end of movement at which muscle activity decreased to baseline levels (off-set).

Pre- (concentric) and post- (eccentric) movement aEMG activity of each muscle was determined from the 500 ms prior to, or after the ROM, for the concentric and eccentric actions, respectively, and was normalized to EMG of the respective muscle during MVC.

Data Reduction

Subjects were unable to accurately produce the faster desired velocities despite best effort and the use of the velocity templates. Only 3 subjects were able to perform the concentric movements, and only 2 subjects were able to perform the eccentric movements, at an average velocity faster than 1.05 rad · s^{-1}. Therefore, the velocity for each of the different conditions was examined on the basis of actual average velocity obtained during the trials of each condition. After initial examination of the obtained average velocities for the different conditions, three different velocity conditions were chosen for further examination. Conditions were chosen based on the actual average velocity, regardless of the attempted velocity, and thus the average velocity of the concentric and eccentric conditions used for further analysis were matched as closely as possible between actions. The final average velocities were 0.17, 0.52 and 0.79 rad · s^{-1}, and 0.17, 0.50 and 0.74 rad · s^{-1} for the concentric and eccentric velocities, respectively. The 3 velocities of each contraction were significantly different ($P < 0.001$) but the slowest (V1), medium (V2) and fastest (V3) velocity of each contraction were not significantly different between concentric and eccentric contractions. The movement times (ms) were; V1 - 3128 vs. 3264, V2 – 1031 vs. 992 V3 – 640 vs. 700, for the concentric and eccentric contractions, respectively.

Statistical Analysis

Two-factor analysis of variance (ANOVA) was used to compare the actual velocities obtained during the different attempted velocities for both muscle actions. The effects of muscle action (concentric versus eccentric), velocity (V1, V2,V3) and muscle (TA, SOL, LG, MG) on the EMG variables of aEMG, pEMG and time to pEMG, were evaluated using separate four-way ANOVA with repeated measures for each variable. Muscle activity on/offset and pre-/post-movement data were both analyzed with separate two-factor ANOVA with a repeated-measures design (velocity x muscle) for each muscle action. To examine the pattern of muscle activation, a four-factor repeated measures ANOVA design was used (action x velocity x muscle x bin). Agonist and antagonist muscles were examined separately as comparisons between the bin values of agonist and antagonists were not of interest. The kinematic data were evaluated with a two-factor ANOVA (action x velocity) repeated-measures design and the analysis of the movement patterns and force used a three-factor repeated measures ANOVA design (action x velocity x bin). A between factor of sex was initially included in all analyses but this factor did not demonstrate any significant main effects and thus the groups were collapsed across sex. The level of significance was set at $P < 0.05$ for all analyses and Newman-Keuls post hoc tests were used to locate differences when significant interactions were found. All results are reported as means and SE unless otherwise stated. The statistical software program used was Statistica (Statsoft, OK).

Results

Velocity and Acceleration

Average velocity across the 10 bins (significant velocity by bin interaction, $P < 0.0001$) is shown in Fig. A-2a. There was no main effect for action or significant interaction of action by velocity by bin, indicating that the velocity pattern throughout the movement followed the same pattern during both actions. No statistically significant differences in velocity occurred between all bins at V1 and the middle bins of V2, indicating that constant velocity was a feature of concentric and eccentric actions at the slower velocities (V1 and V2). Average velocity of the first and last bins did not differ between the 3 velocities but increased average velocity of the total movement was reflected by significant differences between corresponding bins at different velocities (V1-V3, bins 2-9, V1-V2, bins 2-8, and V2-V3, bins 3-9, $P < 0.05$). There was no difference in velocity between concentric and eccentric contractions but peak velocity increased across the velocity conditions (main effect for velocity only, $P < 0.0001$).

Muscle action did not affect the acceleration pattern (no main effect or interactions with action and the other factors). The acceleration profiles of V1 and V2 supported the finding of constant velocity throughout the movement, as acceleration across all bins for V1 and V2 was close to zero (Fig. A-2b). The muscle actions during V3 were performed more ballistically, as indicated by the bell shaped velocity curve and a biphasic (positive-negative) acceleration profile with the average bin acceleration of the earlier bins (1-5) all greater than the later bins (6-10) (all between bin comparisons $P < 0.01$) (Fig. A-2a,b).

Although there was no main effect for sex, there was a significant ($P < 0.001$) 4-way interaction between sex x action x velocity x bin (results not shown). This interaction was attributed mainly to a difference in the performance of V3. During concentric V3 females demonstrated higher average bin velocities in the earlier bins while males reached higher velocities in the middle bins. During eccentric V3, females had a plateau in velocity across the middle bins while the males showed an increase in velocity to a peak in bins 5 followed by decreased velocity to bin 10.

Peak Velocity

Muscle action did not influence peak velocity but peak velocity increased during the velocity conditions (main effect for velocity only, $P < 0.001$, no significant main effect for action or significant interaction of action and velocity). Peak velocities (SE) were; V1- 0.38 (0.02), vs. 0.41 (0.03), V2 - 1.07 (0.08) vs. 1.11 (0.08) and V3 - 1.50 (0.06) vs. 1.79 (0.1) rad $\cdot$ s^{-1} for the concentric and eccentric actions, respectively.

As with velocity, with the consideration of acceleration, there was no main effect for sex, but there was significant ($P < 0.05$) interaction between sex, action and bin as well as between sex and bin. As the effects of velocity were of primary interest, these results were not considered to be central to the principal findings and are not presented.

Kinetic Pattern

There was a significant ($P < 0.01$) three-way interaction of action x velocity x bin for average bin force (Fig. A-3). Although the external load was the same during both muscle actions, the load, all concentric average bin force values were approximately two-fold greater ($P < 0.001$) than eccentric values. This may have been due the experimental setup and location of the force transducer. Velocity had no influence on average force of corresponding bins between concentric V1-V2 and V2 -V3 and between all eccentric velocities. Only bin 2 between concentric V1 - V3 differed ($P < 0.05$). Within each muscle action at each velocity, there were no significant differences in average force for all bins at concentric V1 and V2 and at all eccentric velocities. Significant differences in average force of the bins during a condition were only found during concentric V3, with earlier bins demonstrating larger forces than later bins (bin 2 > bins 7-10, bin 3 > bins 8-10, bins 4 and 5 > bins 9, 10, all $P < 0.05$-0.01). Within all other muscle action and velocity conditions, all bin force values did not differ statistically, indicating even tension development throughout these movements.

Muscle action did not influence peak velocity but peak velocity increased during the velocity conditions (main effect for velocity only, $P < 0.001$, no significant main effect for action or significant interaction of action and velocity).

Average EMG

Fig. A-4 shows the aEMG data for all subjects at all velocities. Significant relationships between velocity normalized to peak average velocity and aEMG were found at all concentric velocities but there was an absence of significant relationships with eccentric actions. Fig. A-5 shows the aEMG response of all muscles at all velocities. There was a significant ($P < 0.001$) 3-way interaction between muscle action, velocity and muscle. All agonist muscles demonstrated a similar pattern of response with aEMG greater during concentric actions than eccentric actions at all velocities (all $P < 0.01$). As velocity increased during concentric actions, aEMG also increased (all $P < 0.05$ - $P < 0.01$). However, aEMG of the 3 agonists did not change with eccentric velocity. Although there were no differences in muscle activation between the agonist muscles during concentric V1, LG was the most active at V2 and V3 ($P < 0.05$). Muscle activation during eccentric actions differed in that MG was the most active of the agonists at all eccentric velocities ($P < 0.05$).

aEMG of antagonist TA exhibited a different response than the agonist muscles. aEMG did not differ between actions at each velocity, as it did with the agonists. Concentric velocity had no influence on TA aEMG but during eccentric actions, TA aEMG increased with velocity ($P < 0.01$). TA aEMG was lower (all $P < 0.001$ at all velocities) than all of the agonist muscles during concentric but not eccentric actions. During eccentric actions, SOL aEMG was lower ($P < 0.05$) than that of TA at V3, and aEMG of MG was greater ($P < 0.01$) than that of TA at V1 and V2.

Average EMG of Bins

Fig. A-6 shows the pattern of aEMG over the movement. There was a significant ($P < 0.001$) four-way interaction between action, velocity, muscle and bin for the agonist muscles. During concentric actions, all agonists demonstrated an increase in aEMG from bin 1 to 10 (all $P < 0.0001$). In general, during V1, aEMG of all agonists increased between successive bins, with aEMG of each bin greater than the earlier bins ($P < 0.05$). During V2 and V3, aEMG demonstrated a plateau with no further increase in bin aEMG after bin 6 to 7, with no difference between bin aEMG of each agonists in these later bins. This leveling off occurred even earlier in SOL (Bin 4) during V3. Concentric V1 had little influence on aEMG of the bins between the agonists but as velocity increased, LG became more active than SOL in the later bins (bins 6-10, all $P < 0.02$). These results indicate that as velocity increased, SOL became relatively less active while LG became more active, especially in the later bins. Velocity did not change pattern of activation of MG, other than an overall increase in aEMG with velocity.

The pattern of activation of the agonists over the bins during eccentric actions at each velocity was opposite to that during concentric actions, with decreased aEMG from bin 1 to 10 (all $P < 0.001$). Overall MG was the most active in all bins (MG > SOL, all bins all $P < 0.001$, MG > LG, bins 3-10 all $P < 0.01$), SOL was the least active, and aEMG of LG was between MG and SOL (LG > SOL, bins 1-4, $P < 0.05$). Velocity did not influence the activation pattern, with no difference in aEMG between the corresponding bins of different velocities.

Velocity did not influence the activation pattern of TA but muscle action did (significant interaction of action by bin, $P < 0.001$, although the 3-way interaction of action x velocity x bin approached significance, $P = 0.06$). During concentric actions, collapsed across all velocities, TA demonstrated the same pattern as the agonists during concentric actions, with an increase in bin aEMG throughout the ROM, with bin 1 < bin 10 ($P < .001$). However, during eccentric actions, the bin aEMG displayed a similar pattern as during concentric actions, or the opposite pattern found with the agonists during eccentric actions. Thus activity of TA increased throughout the ROM during both actions, collapsed across velocity, with activity in the earlier bins less than that of the later bins (concentric bins 1-6 < bins 8-10, $P < 0.01$; eccentric bins 1-5 < bins 8-10, $P < 0.05$).

Peak EMG

The pattern of response of pEMG was similar to that of aEMG. There was a significant three-way interaction between action, velocity and muscle for pEMG ($P < 0.01$, Fig. A-7). Concentric pEMG was significantly greater than eccentric pEMG at all corresponding velocities for all agonist muscles (all $P < 0.01$). Concentric velocity influenced the pEMG of LG with peak values increasing with velocity ($P < 0.05$) but velocity had no effect on SOL and MG pEMG during all concentric velocities. The pEMG of LG at V2 and V3 was greater (all $P < 0.05$) than the pEMG of both SOL and MG. During eccentric actions, velocity had no effect on pEMG of the agonists and there was no difference in pEMG between the agonist muscles (this response differed from that of aEMG).

There was no difference in pEMG of antagonist TA between muscle actions at V1 and V2 but during V3, eccentric pEMG was greater than concentric pEMG ($P < 0.05$). Velocity had no influence on pEMG of TA during concentric actions but during eccentric actions, pEMG increased ($P < 0.01$) with velocity. The pEMG of TA was less than all agonists (all $P < 0.001$) during all concentric conditions but with the increase in pEMG of TA with velocity during eccentric actions, pEMG of TA, LG and MG not differ during eccentric V2 and V3.

Temporal Pattern of Peak EMG

There was a significant ($P < 0.001$) three-way interaction between action, velocity and muscle for time of pEMG (Fig. A-8). All agonist muscles responded the same with the time of pEMG occurring later at all velocities during concentric actions than during eccentric actions (all $P < 0.001$). As velocity increased, the pEMG occurred earlier ($P < 0.001$) during concentric but not eccentric actions. Similar to the agonists, the time of pEMG of the TA occurred significantly later during concentric actions than during eccentric actions but only during V2 and V3. TA demonstrated the opposite response of the agonists with respect to velocity during each action. Velocity had no effect on the time at which pEMG occurred during concentric actions, but pEMG occurred earlier as velocity increased during eccentric actions ($P < 0.05$). At each concentric velocity, the time at which pEMG occurred for all 4 muscles examined did not differ except for pEMG of TA occurred later than that of SOL during V3 ($P < 0.05$). However, during eccentric actions at all velocities, the time of pEMG for the antagonist TA occurred significantly later than that of the agonists (Fig. A-9).

Muscle Activity On/Offset

Interaction between velocity and muscle was significant ($P < 0.001$) for muscle activity onset (Fig. A-9). All 4 muscles behaved similarly with respect to velocity, with muscle activity onset occurring earlier as velocity increased ($P < 0.001$). SOL activity was initiated earlier than LG and MG during V1 ($P < 0.01$) but the muscles did not differ at the faster velocities. TA onset began later after the start of movement during the slowest velocity but did not differ from the agonists at the other velocities.

Interaction between velocity and muscle was not significant ($P = 0.05$) for muscle activity offset but main effects for both velocity and muscle were significant (both, $P < 0.001$) (Fig. A-10). The effect of velocity on all muscles combined was for the muscles to stay active longer after the end of the ROM as velocity increased (all velocities, $P < 0.01$). Collapsed across velocities, TA stayed active longer after the end of the ROM than the agonists.

Pre/Post Movement Muscle Activity

Muscle activity of all muscles combined and for the 500 ms period both before the initiation of movement and after the end of movement was influenced by velocity (significant main effect for velocity, $P < 0.01$). aEMG was greater at V3 (V1-V3, $P < 0.05$, V2-V3, $P < 0.01$) and did not differ between V1 and V2. In general, at all velocities (significant interaction of action x muscle, $P < 0.05$), there was no difference between pre- and post-movement activation of the agonists but post-movement activation of TA was 3-fold higher ($P < 0.01$) than before movement. Pre-movement activation did not differ between all muscles but after the end of movement, both TA and MG were more active (both $P < 0.01$) than SOL and LG. Fig. A-10 shows the data for all muscles for both actions. There were no other significant main effects or interactions.

Discussion

The main findings of this study were that muscle action and velocity had a differential effect on muscle activation such that: 1) a velocity-dependant muscle activation relationship occurred during concentric but not eccentric actions that was similar across all muscles of the triceps surae; 2) specific differences in activation patterns occurred with the synergist triceps surae muscle group; 3) the differences in muscle activation between actions was partially offset with comparisons at similar muscle length, and; 4) the behaviour of the antagonist TA was different than the agonist triceps surae muscles.

Muscle activation (both peak and average) of all agonist muscles was found to be significantly greater during concentric actions than eccentric actions, reflected by both aEMG and pEMG. Furthermore, velocity influenced muscle activation during concentric actions but not eccentric actions, with muscle activation increasing with concentric velocity but remaining constant with all eccentric actions with the same absolute load. This substantiates the findings regarding the gastrocnemius of the classic investigation of Bigland and Lippold (1954) shown almost 50 years ago, and extends the relationship to not only to all three heads of the triceps surae, but also to a greater submaximal load used with the current investigation. Muscle activation of the soleus muscle only during concentric and eccentric plantar flexion with a fixed submaximal load of approximately 15-20% of MVC was reported by Romano and Schieppati (1987). They reported that EMG of the soleus increased with concentric velocity but decreased with eccentric velocity. As well, EMG increased throughout the movement during concentric actions with an opposite pattern found during eccentric actions. The data of the soleus during the current investigation partially match these observations, and also apply to the LG and MG muscles. However, the finding of an inverse relationship between soleus activation and eccentric velocity is in contrast to the current finding of similar EMG amplitude of all agonist muscle irrespective of velocity. This discrepancy in findings is difficult to explain, but may be related to a difference in quantification of the results. Despite this discrepancy, the findings of the current study regarding muscle activation during concentric and eccentric muscle actions using a submaximal load corroborate and expand on previous reports regarding the gastrocnemious (Bigland &Lippold; 1954) and soleus (Romano & Schippati, 1987) muscles.

During dynamic concentric and eccentric actions with a submaximal load, motor units have demonstrated differential behavior between the muscle actions (Howell et al., 1995; Nardone et al., 1989; Tax et al., 1989; Theeuwen et al., 1994) and may explain why differences occur in EMG between the actions. Greater motor unit firing frequency (Tax et al., 1989) and lower recruitment thresholds for motor units (Tax et al., 1989; Theeuwen et al., 1994) during concentric actions may result in overall increased motor unit activity, as assessed by surface EMG.

The finding of a difference in the pattern of activation of the synergist agonist muscles throughout the movement with increased velocity during concentric actions of the current investigation was interesting. While the aEMG of both gastrocnemius heads increased with muscle shortening, SOL demonstrated a plateau in muscle activation midway through the movement. A similar observation was seen in the data of Romano and Schieppati (1987). Together, these observations suggest a velocity dependant shift between synergist muscles. This shift may be related to the motor unit (fibre type) composition of individual muscles of the triceps surae (Alway et al., 1989; Johnson et al., 1973). Thus, with increased movement velocity, the LG and MG, with a greater proportion of faster motor units, became relatively more active.

An interesting finding was the alteration in activation of the two heads of the gastrocnemius between actions. LG activation was significantly greater than the other synergist muscles during concentric actions, while during eccentric actions, MG activation was significantly greater than the other muscles. This alteration in synergist muscle activation, especially of muscle of similar fibre type composition and architecture, dependent on muscle action, may represent a mechanism by which fatigue can be minimized in synergist muscles. A similar muscle action dependant alteration between synergist muscles of the elbow flexors has also been previously reported (Nakazawa et al., 1993; Tax et al., 1990).

The aEMG increased throughout the ROM during concentric actions. Thus, aEMG increased as the muscle shortened. The opposite pattern was found during eccentric actions with decreased aEMG from the beginning to end of the movement. In both actions, aEMG was greater at shorter muscle lengths and was lower at longer muscle lengths, as suggested previously (Romano & Schieppati, 1987). This difference in activation at different muscle lengths may be related to the length-tension relationship of muscle. There may not have been optimal tension generation through cross-bridge formation at the shorter muscle lengths and thus relatively greater muscle activation was required in order to maintain the level of tension to over come the external load. The muscle fibres were likely operating on the ascending limb of the tension-length. While this can explain the pattern of aEMG throughout the ROM, it does not explain the significantly lower levels of activation during the eccentric actions.

It has been suggested that motor unit activation during very slow eccentric actions, even during very slow movements with a submaximal load, may not follow Henneman's size principle, and high-threshold motor units may be preferentially recruited during eccentric muscle actions (Howell et al., 1995; Karapondo, et al., 1993; Nardone et al., 1989). In addition, a shift in activity between muscles comprising predominately low-threshold motor units (soleus) to those with a greater proportion of high-threshold motor units (gastrocnemius) has been reported to occur during eccentric actions (Nardone & Shieppati, 1988). The current results do provide some support for this finding. While it was found that the MG was significantly more active than SOL during eccentric actions, SOL was not relatively more active during concentric actions. As well, SOL did become relatively less active at the faster eccentric velocities. While a similar muscle action dependant alteration between synergist muscles of the elbow flexors has been previously reported (Nakazawa et al., 1993; Tax et al., 1990), the muscles involved in these investigations did not greatly differ in composition.

During eccentric actions, antagonist activation increased in the later part of the muscle actions. This increased activity throughout the lengthening actions suggests that the role of the antagonist is to 'brake' or slow down the movement. Although it seems as though this strategy would be important during rapid movements with greater changes in velocity and acceleration, average velocity did not alter antagonist activation. It may also be that TA activity is used to exert overall control of movement, especially if fewer motor units are active during eccentric actions. Muscle action also did not influence antagonist activation. This suggests that that there is one control strategy during both actions, even during very slow movements, and that the role of antagonist activation is to 'brake' or slow down the movement at the end of the ROM. However, it is difficult to explain why TA activation did not change with velocity, as the slowest velocity displayed minimal changes in velocity and acceleration.

This finding of the antagonist being active during PF is in contrast to other investigations. No antagonist activation was found in the experiments of Nardone and co-workers (Nardone & Schieppati, 1988; Nardone et al., 1989) and that of Tamaki et al. (1997), despite similar experimental setups. This inconsistency in findings may be related to the load used in these studies was less than that of approximately 30% MVC of the current investigation. This suggests that load may influence antagonist activation and further investigation is required. However, little to no antagonist activation has been reported to occur with elbow flexion, even with low to moderate load (Abbruzzese et al., 1994; Nakazawa et al., 1994). In addition, antagonist activation did occur in isokinetic dorsiflexion (Behm & Sale, 1993). This suggest that there is a threshold load or level for antagonist activation but further investigation is required to clarify the role of antagonist co-activation.

Examination of the temporal patterns of EMG timing during concentric and eccentric muscle actions shows a reduction in the time to pEMG with increased average velocity. In contrast, there was no variation in the time to pEMG of the triceps surae muscles during eccentric actions. This suggests that with concentric actions there is not only EMG amplitude modulation, but also a corresponding difference in the timing of activation.

Gender differences in neural control of movement has not received a great deal of attention, and gender did not influence the results of the current investigation. On theoretical grounds, a differential response between gender regarding neural control of voluntary movement is not expected. However, this has not been extensively addressed in the literature, as the majority of studies have used only one gender, usually males. Interestingly, gender differences in antagonist muscle activation have been observed during slow movements (Behm & Sale, 1996; Solomonow et al., 1988). In addition, gender differences in EMG parameters of the antagonist triceps brachii during rapid elbow flexion exercise have also been reported (Ives et al., 1993). Further investigation into gender differences in neural activation, especially regarding antagonist activation is required.

In summary, the results of this investigation support the suggestion that the nervous system utilizes different activation strategies for concentric and eccentric muscle actions (Enoka, 1996). Despite these different muscle action dependant activation strategies, neural control of movement is likely based on evaluation of the task as a whole. Thus while differences in activity may occur with specific muscles, and may differ with muscle action, the end result is generation of torque around a specific joint and movement. Subtle shifts in activity across different muscles can be expected to occur for motor control.

REFERENCES

Abbruzzese G, Morena M, Spadavecchia L & Schieppati M (1994) Response of arm flexor muscles to magnetic and electrical brain stimulation during shortening and lengthening tasks in man. J Physiol (Lond) 481(2): 499-507.

Alway SE, Hughson RL, Green HJ & Patla AE (1988) Human tibialis anterior contractile responses following fatiguing exercise with and without beta-adrenoceptor blockade. Clin Physiol 1998 Jun 8: 215-225.

Alway SE, MacDougall JD & Sale DG (1989) Contractile adaptations in the human triceps surae after isometric exercise. J Appl Physiol 66: 2725-2732.

Aura O & Komi PV (1986) Mechanical efficiency of pure positive and pure negative work with special reference to the work intensity. Int J Sports Med. 7: 44-49.

Behm DG & Sale DG (1993) Intended rather than actual movement velocity determines velocity- specific training response. J Appl Physiol 74: 359-368.

Behm DG & Sale DG (1996) Influence of velocity on agonist and antagonist activation in concentric dorsiflexion muscle actions. Can J Appl Physiol 21(5): 402-416.

Bigland B & Lippold OC (1954) The relation between force, velocity and integrated electrical activity in human muscles. J Physiol (Lond) 123: 214-224.

Dalton PA & Stokes MJ (1991) Acoustic myography reflects force changes during dynamic concentric and eccentric contractions of the human biceps brachii muscle. Eur J Appl Physiol Occup Physiol 63: 412-416.

Donne B & Luckwill RG (1996) Co-activation of quadriceps and hamstring muscles during concentric and eccentric isokinetic exercise. Isokinetics Exerc Sci 6(1): 21-26.

Enoka RM (1996) Eccentric contractions require unique activation strategies by the nervous system. J Appl Physiol 81: 2339-2346.

Fang Y, Siemionow V, Sahgal V, Xiong F & Yue GH (2001) Greater movement-related cortical potential during human eccentric versus concentric muscle contractions. J Neurophysiol 86: 1764-1772.

Gillies E, Zehr EP, Haridas C & Sugajima Y (2000) Muscle activation during lengthening and shortening plantar flexion of varying duration with a submaximal load. Can J Appl Physiol 25: 374.

Gulling L K, Lephart S M, Stone D A, Irrgang JJ & Pincivero D M (1996) The effects of patellar bracing on quadriceps EMG activity during isokinetic exercise. Isokinetics Exerc Sci 6(2): 133-138.

Howell JN, Fuglevand AJ, Walsh ML & Bigland-Ritchie B (1995) Motor unit activity during isometric and concentric-eccentric contractions of the human first dorsal interosseus muscle. J Neurophysiol 72(2): 901-904.

Ives JC, Kroll WP & Bultman LL (1993) Rapid movement kinematic and electromyographic control characteristics in males and females. Res Q 64(3): 274-283.

Karapondo et al. 1993

Kawakami Y, Ichinose Y & Fukunaga T (1998) Architectural and functional features of human triceps surae muscles during contraction. J Appl Physiol 85: 398-404.

Kellis E & Baltzopoulos V (1998) Muscle activation differences between eccentric and concentric isokinetic exercise. Med Sci Sports Exerc 30(11):1616-1623.

Komi PV, Linnamo VA, Ventoinen PS & Sillanpaa M (2000) Force and EMG power spectrum during eccentric and concentric actions. Med Sci Sports Exerc 32(10): 1757-1762.

McHugh MP, Tyler TF, Greenberg SC & Gleim GW (2002). Differences in activation patterns between eccentric and concentric quadriceps contractions. J Sports Sci (20): 83-91.

Nakazawa K, Kawakami Y, Fukunaga T, Yano H & Miyashita M (1993) Differences in activation patterns in elbow flexor muscles during isometric, concentric and eccentric contractions. Eur J Appl Physiol Occup Physiol 66: 214-220.

Nakazawa K, Yamamoto SI & Yano H (1997) Short- and long-latency reflex responses during different motor tasks in elbow flexor muscles. Exp Brain Res 116: 20-28.

Nardone A, Romano C & Schieppati M (1989) Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscles. J Physiol (Lond) 409: 451-471.

Nardone A & Schieppati M (1988) Shift of activity from slow to fast muscle during voluntary lengthening contractions of the triceps surae muscles in humans. J Physiol (Lond) 395: 363-381.

Nichols TR (1994) A biomechanical perspective on spinal mechanisms of coordinated muscular action: an architecture principle. Acta Anat 151: 1-13.

Romano C & Schieppati M (1987) Reflex excitability of human soleus motoneurones during voluntary shortening or lengthening contractions. J Physiol (Lond) 390: 271-284.

Solomonow M, Baratta R, Zhou BH & Ambrosia R (1988) Electromyogram coactivation patterns of the elbow antagonist muscles during slow isokinetic movement. Exp Neurol 100: 470-477.

Tamaki H, Kitada K, Akamine T, Sakou T & Kurata H (1997) Electromyogram patterns during plantarflexions at various angular velocities and knee angles in human triceps surae muscles. Eur J Appl Physiol Occup Physiol 75: 1-6.

Tax AA, Denier van der Gon JJ, Gielen CC & Kleyne M (1990) Differences in central control of m. biceps brachii in movement tasks and force tasks. Exp Brain Res 79: 138-142.

Westing SH, Cresswell AG & Thorstensson A (1991) Muscle activation during maximal voluntary eccentric and concentric knee extension. Eur J Appl Physiol 1991 62: 104-108.

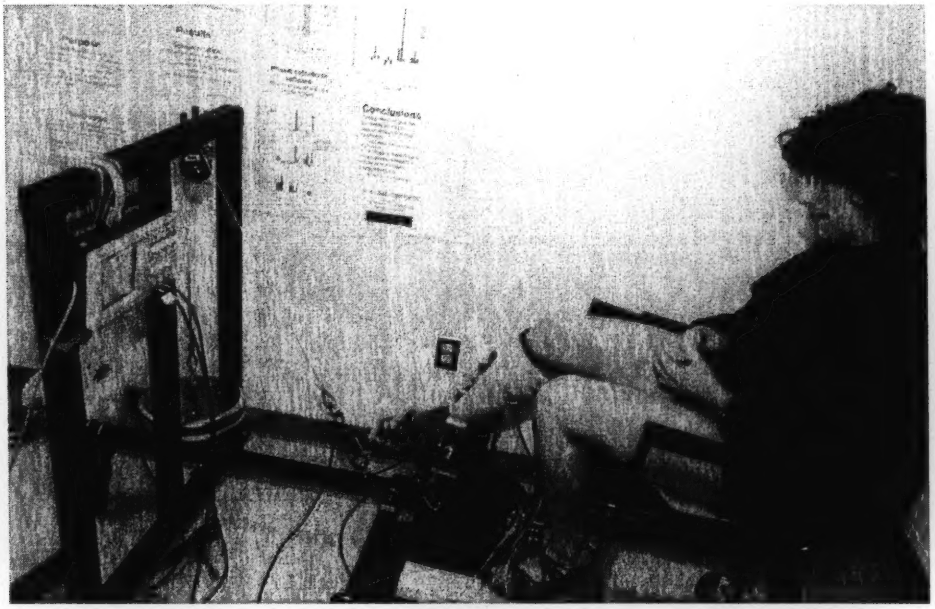
Conclusions

Figure A-1. Experimental setup.

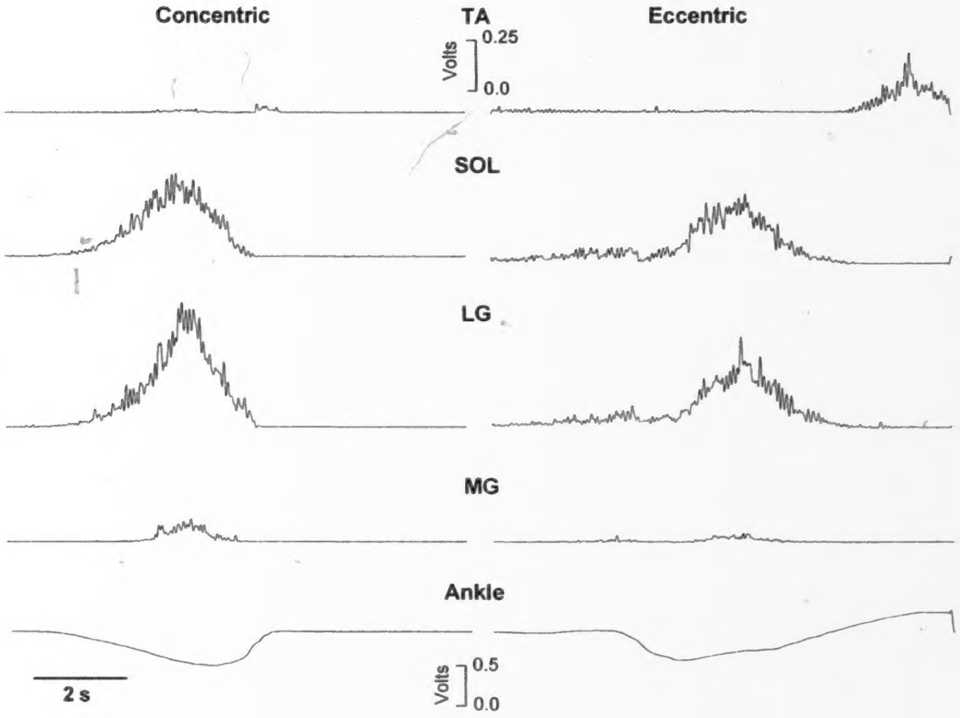

Figure A-2. Sample data for a single subject performing concentric and eccentric contractions of the triceps surae muscles at the slowest performance velocity (V1). Abbreviations: TA, tibialis anterior; SOL, soleus; LG, lateral gastrocnemius; MG, medial gastrocnemius.

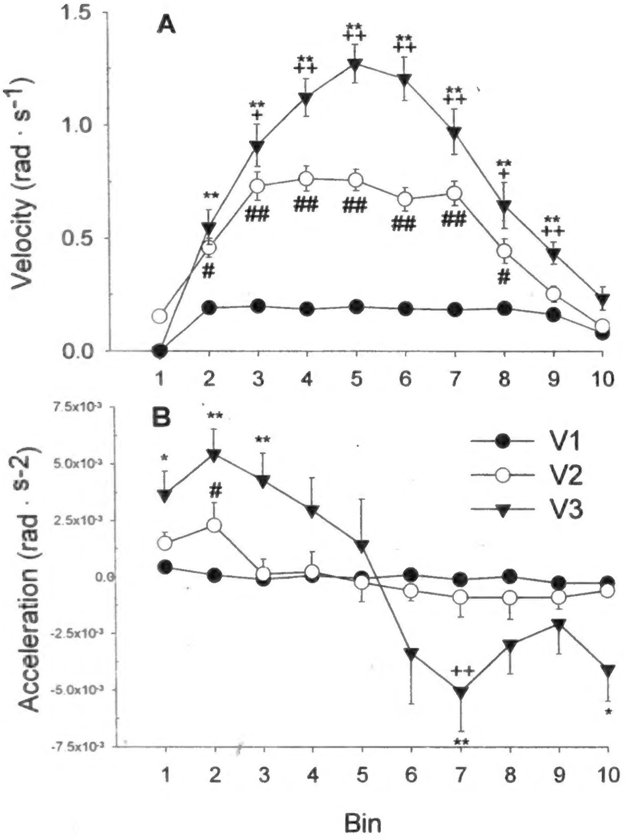

Figure A-3. Kinematic data of velocity (A) and acceleration (B) during the 3 velocity conditions.

V1 is the slowest velocity and V3 is the fastest velocity, with increased average velocity ($P < 0.05$) from V1 To V2 to V3. See text for actual velocities. Note that there was no main effect for action. Significant differences between velocities for the corresponding bin are indicated as: V1-V2; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$; V2-V3; $^{+}P < 0.05$, $^{++}P < 0.01$. Significant differences between adjacent bins are indicated as; * $P < 0.05$, ** $P < 0.01$. Values are bin means and SE.

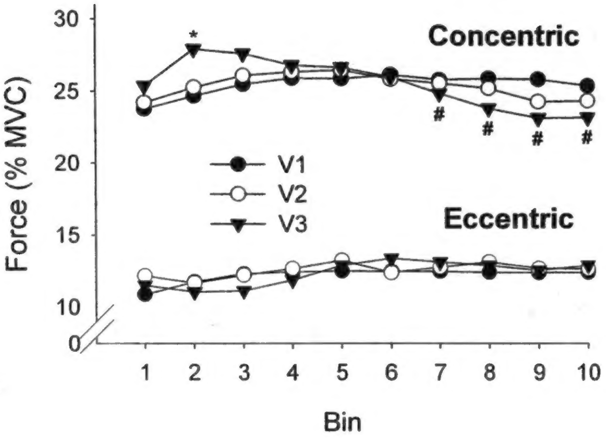

Figure A-4. Average bin force during concentric and eccentric movements at all velocities.
All concentric bin force values were significantly different (P < 0.01) from all eccentric bins. There were no significant differences in force of all bins at concentric V1 and V2 and all eccentric velocities. # During concentric V3, significant differences (P < 0.01) occurred between: bin 2 & bins 7-10; bin 3 & bins 8-10; bins 4-5 and bins 9-10.
* Significant difference (P < 0.05) between bins of concentric V1 and V3. All other comparisons between velocities of each action were not significant. Average velocity increases from V1 to V2 to V3. Error bars are not shown for clarity.

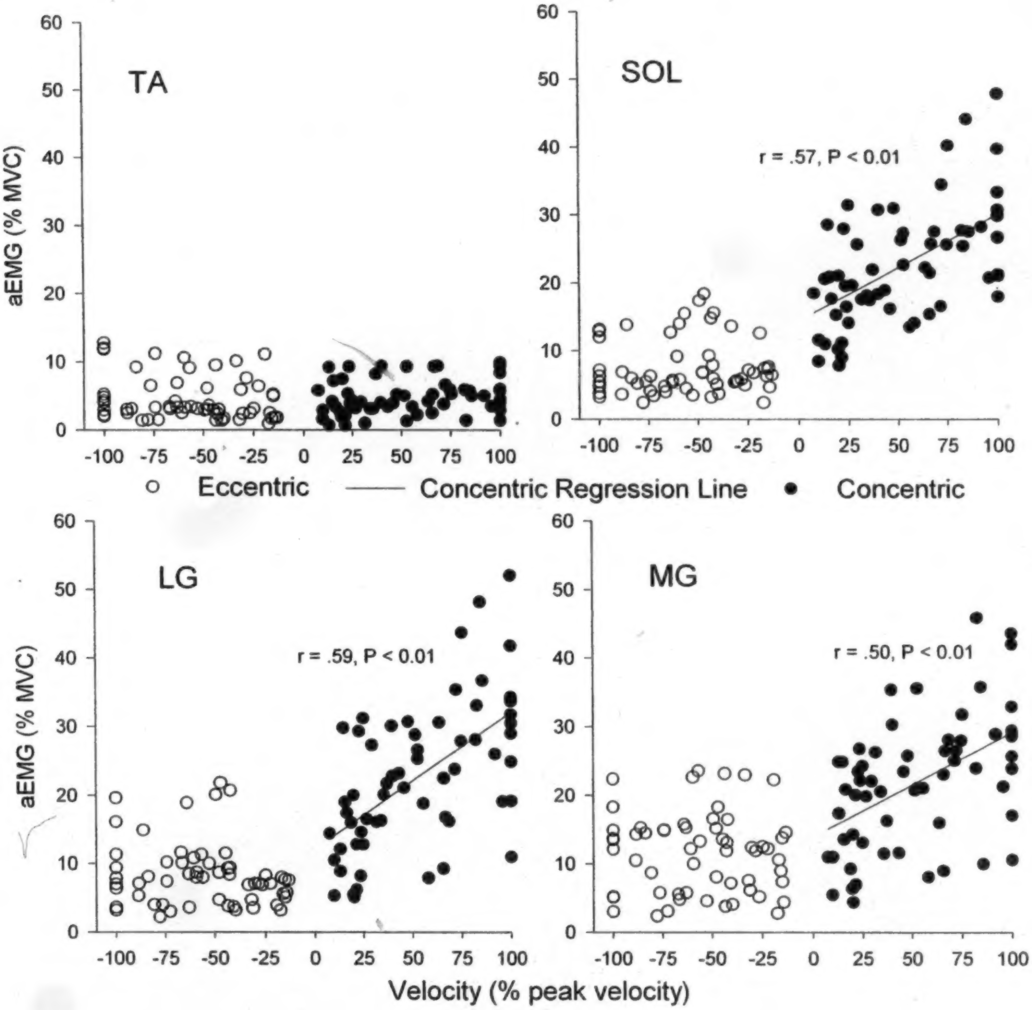

Figure A-5. Relationship between normalized velocity and average EMG for both muscle action conditions and across all muscles.

Values have been normalized to the peak average velocity and the maximal isometric contraction for velocity and EMG, respectively, and are given as percentages. Significant ($P < 0.05$) relationships between EMG amplitude and velocity are indicated by the plotted regression lines. Note the absence of any significant regressions during eccentric actions. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2.

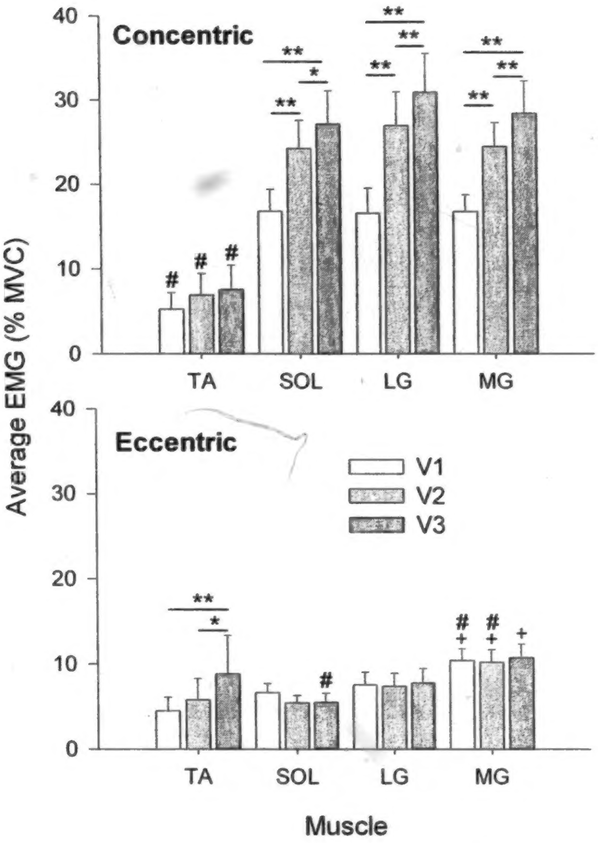

Figure A-6. Average EMG during both muscle actions and across all muscles. aEMG of SOL, LG and MG at all velocities was significantly different (all comparisons, P < 0.01) between concentric and eccentric muscle contractions. + indicates significant differences (P < 0.05) to other agonists at the same velocity and contraction. # indicates significant differences (P < 0.01) between TA and all agonists at the same velocity (concentric) or TA and SOL or MG (eccentric). Significant differences between velocities are indicated with a horizontal line as follows: * P < 0.05 and ** P < 0.01. All values are mean and SE. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2.

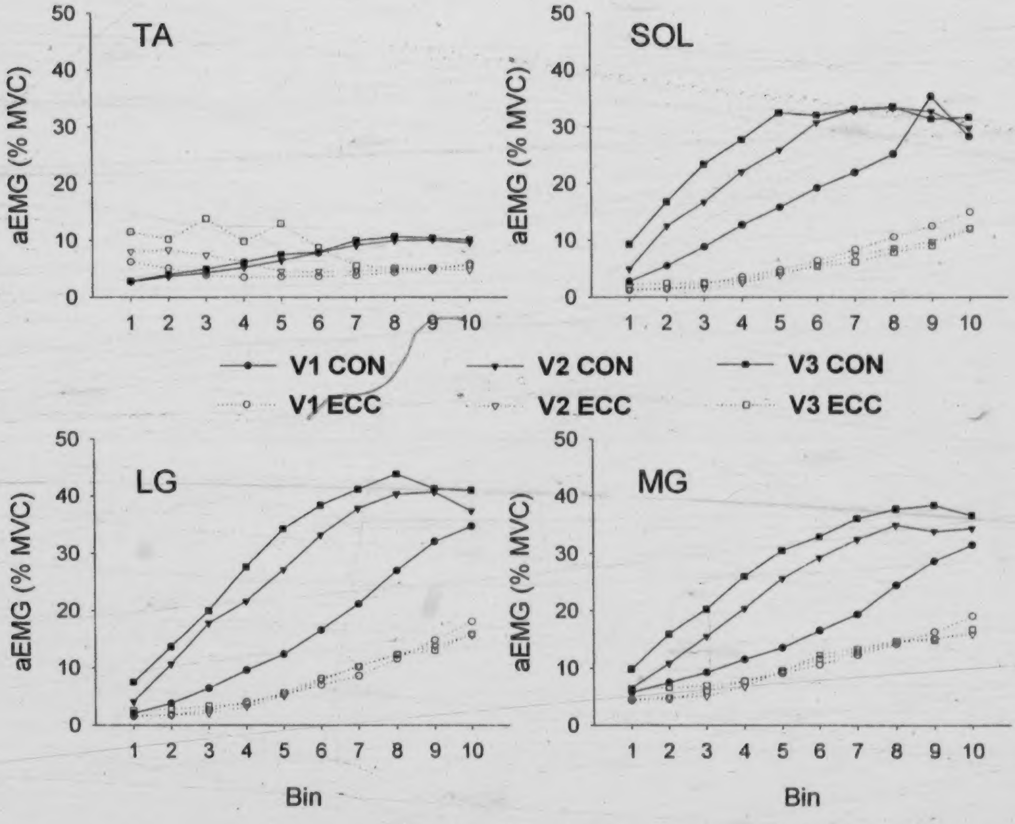

Figure A-7. Normalized average EMG for each movement bin for all muscles during concentric (CON) and eccentric (ECC) muscle contractions.

EMG has been normalized to the maximum isometric value and data are given as percentages. Error bars are not shown for clarity. All values are means. Note that the bin values for both CON and ECC muscle actions are displayed at similar muscle lengths. Thus the bin values are from longer to shorter muscle lengths with shortest muscle lengths at the end of the CON and at the beginning of ECC muscle actions (plantarflexion increases from left to right). Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2.

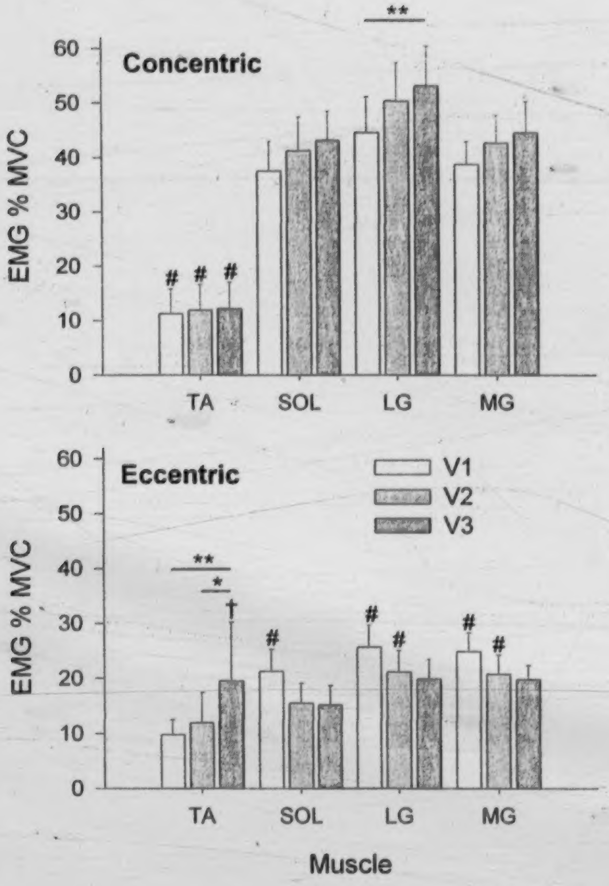

Figure A-8. Peak EMG of all muscles during concentric and eccentric muscle actions at all velocities.

Peak EMG of all agonists was significantly different (all comparisons, $P < 0.01$) between concentric and eccentric muscle actions at all velocities. + indicates significant differences ($P < 0.01$) to other agonists at the same velocity and action. # indicates significant differences ($P < 0.01$) between TA and all agonists at the same velocity (concentric) or TA and the indicated agonist (eccentric). Significant differences between velocities are indicated as * $P < 0.05$ and ** $P < 0.01$. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2. All values are mean and SE.

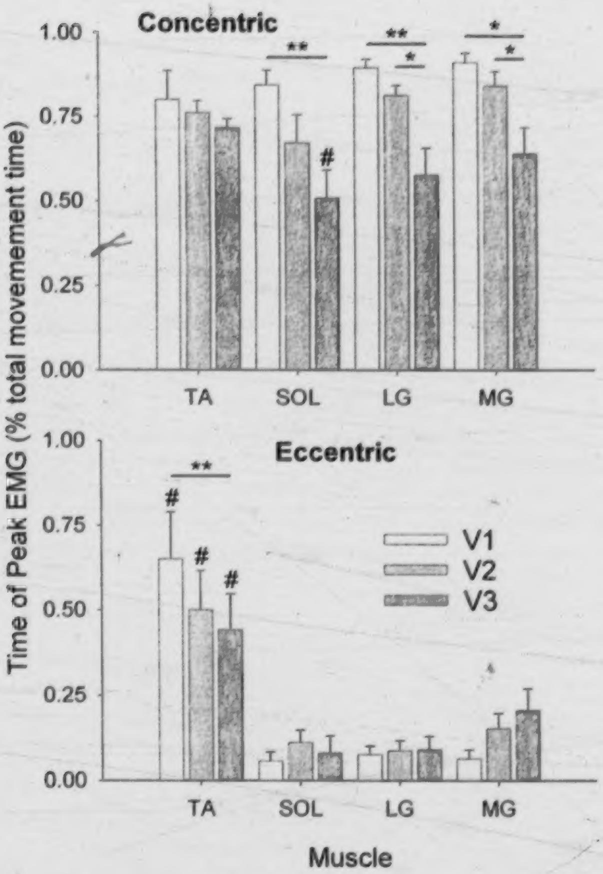

Figure A-9. Time to peak EMG normalized to movement time for all velocity and muscle action conditions.

Values are expressed as percentages of the total movement time for each velocity condition. The time to pEMG of all 4 muscles occurred significantly later (all $P < 0.01$) during concentric than during eccentric actions. # indicates significant differences ($P < 0.05$) between TA and SOL (concentric) or TA and all agonists (eccentric) at the same velocity. Significant differences between velocities are indicated with a horizontal line as follows: * $P < 0.05$ and ** $P < 0.01$. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2. All values are mean and SE.

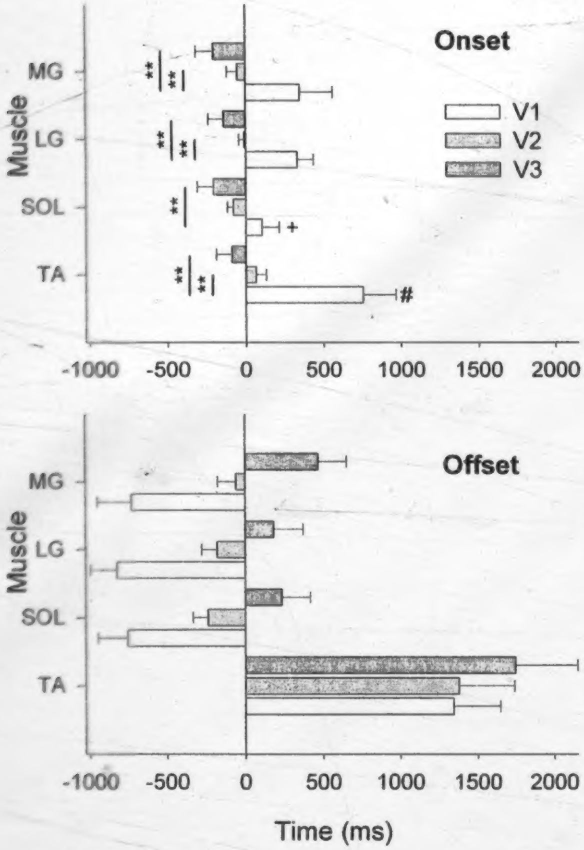

Figure A-10. Muscle activity on/offset.
Negative values indicate that activity began before concentric movement began (onset) or ended before eccentric movement ended (offset). + indicates a significant difference ($P < 0.01$) to other agonists at the same velocity and action. # indicates significant differences ($P < 0.01$) between TA and all agonists at the same velocity. Significant differences between velocities are indicated as ** $P < 0.01$. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2. All values are mean and SE.

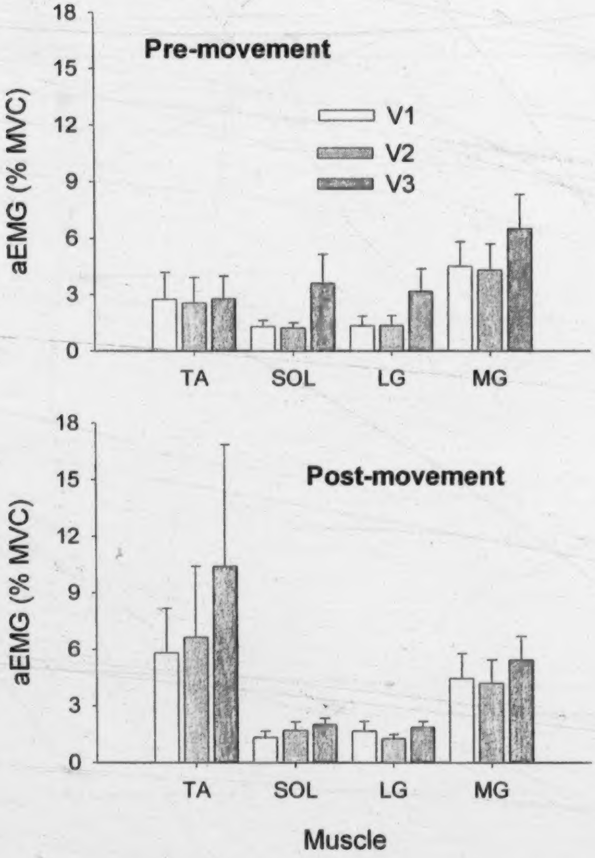

Figure A-11. Pre/post-movement muscle activation.
There were significant main effects for velocity and for muscle action and muscle. Overall, muscle activity increased with velocity. Pre-movement activation of all muscles did not differ but after movement, TA and MG were significantly different ($P < 0.01$) than SOL and LG. TA activation was significantly different ($P < 0.01$) pre and post movement while the agonists did not differ in activation before and after the movement. Average velocity increases from V1 to V2 to V3. Abbreviations are as in Figure A-2. All values are mean and SE.

APPENDIX B DIETARY INTAKE RECORDS FOR ALL RECORDING PERIODS

Table B-1. Mean daily caloric intake and macronutrient intake (% of daily caloric intake) of a 3-4 day period measured every 2 weeks throughout the 9 weeks of training.

	Long Concentric					Long Eccentric				
Record	1	2	3	4	5	1	2	3	4	5
Protein (g • kg body mass • day^{-1})	1.3 (0.3)	1.5 (0.3)	1.5 (0.3)	1.2 (0.3)	1.4 (0.4)	1.2 (0.3)	1.5 (0.4)	1.2 (0.3)	1.4 (0.4)	1.3 (0.3)
Protein (% daily C)	14.8 (2.9)	16.7 (5.5)	16.7 (5.3)	14.5 (6.0)	16.1 (6.0)	15.6 (4.3)	17.7 (4.9)	16.2 (5.2)	16.5 (5.4)	15.8 (3.9)
Carbohydrate (% daily C)	60.7 (8.1)	56.4 (8.8)	60.2 (7.7)	65.3 (10.1)	60.8 (7.6)	60.3 (5.0)	55.6 (6.4)	57.3 (7.7)	59.4 (10.7)	58.5 (8.2)
Fat (% daily C)	23.4 (7.2)	26.1 (6.7)	21.5 (6.6)	19.9 (6.3)	21.6 (5.8)	21.9 (5.8)	25.4 (7.2)	25.9 (9.4)	22.8 (8.0)	23.8 (8.4)
Alcohol (% daily C)	1.4 (2.7)	1.2 (1.9)	1.0 (1.9)	1.6 (1.6)	1.3 (2.1)	1.6 (3.1)	1.3 (2.9)	0.7 (1.9)	1.0 (1.6)	1.3 (3.3)
Calories	2218 (374)	2395 (556)	2342 (484)	2293 (324)	2287 (534)	2178 (275)	2295 (349)	2134 (471)	2375 (407)	2266 (332)

All values are mean (SD) for each multi-day recording period. C – Calories. Note that there were no significant differences between groups for any of the variables and between any of the records.

APPENDIX C

PARTICIPANT INFORMATION LETTER AND INFORMED CONSENT

Resistance training with manipulation of concentric and eccentric muscle actions: muscle adaptations.

Participant Information Letter

Primary Investiagtor: Ellen Gillies, M.Sc., Ph.D. candidate 492-5503

Co-Investigators:	Gordon Bell, Ph.D.	Ted Putman, Ph.D.	Tom Martin, M.D., Ph.D.
	492-2018	492-2187	432-0211

Faculty of Physical Education and Recreation
University of Alberta
E 401 Van Vliet Centre
Edmonton, AB T6G 2H9

Purpose: The purpose of this study is to look at the effects of two different resistance (weight) training programs on maximum strength, muscle size, muscle proteins, muscle damage and hormone levels. The training programs will differ in the time to do the lifting and lowering of the weight.

Eligibility Criteria: You are a woman aged 20-40 years with regular menstrual cycles, no current oral contraceptive use, weight training experience, currently doing 1-2 training sessions a week, and familiar with repetition maximum training and testing.

Procedures:

1. You will have your height, weight and age measured. You will also have your body composition measured with the skinfold technique at six different sites on your body (mid-back, top of hip, triceps, abdomen, front and rear thigh). Your weight and body composition will be measured before and after 9 weeks of training.

2. You will complete a questionnaire about your menstrual cycle. This information will be used to describe the participants and to decide when you will start training. Training will start within 3-9 days of the start of your cycle.

3. You will perform strength tests that will assess how much weight you can lift, lower, and lower and lift for 1 repetition (1RM) for 2 exercises (leg press and bench press) (3 tests for each exercise). These tests will be done before you start the training program, and after 4 and 9 weeks of training. You will do a 10 minute warm-up and cool-down of easy cycling and stretching at each test session.

4. You will have a muscle biopsy taken from the outer thigh muscle before and after 9 weeks of training. A physician will do the biopsy procedure. The procedure involves the sterile preparation of the mid-thigh area. Two ml of local anaesthetic will be injected at the biopsy site so that you will not feel any pain. Then a small incision (< 1 cm) through the skin and fascia on the outside portion of your thigh will be made. A

biopsy needle is inserted and a small sample of muscle tissue (≈ 50 mg – about half the size of a tic-tac) is removed by suction. After being frozen, you may report feeling only "pressure" during the removal of the sample. No stitches are required. A steri-strip bandaid will be used to close the cut. One to two days after the procedure, the biopsy site usually feels like a slight "charley horse" with some local bruising. The muscle sample will be used to measure the proportions of different muscle fibre types, fibre size, blood capillaries and damage to the fibres. As well, the quantity of different muscle proteins will be measured.

5. You will have a morning (between 7 and 9 am) blood sample (10 ml) taken from an arm vein. You will not eat or drink anything except water before this test. The blood sample will be taken before and after 9 weeks of training. Your blood will be used to measure a number of different enzymes (creatine kinase and lactate dehydrogenase), hormones (estradiol, progesterone, total testosterone, free testosterone, cortisol, human growth hormone, sex hormone binding globulin, luteinizing hormone and androstenedione), hematocrit and hemoglobin.

6. You will collect your urine for a 24-hour period during weeks 1 and 9 of training. Your urine will be used to measure protein usage and breakdown.

7. You will do a 4-day dietary record during the first week of training. You will eat 1.5 g of protein per kg body weight each day during these 4 days. You are free to make your own food choices, but you will not eat any meat or fish. You will follow the same plan during the last week of training. You will also record your food intake about every 2 weeks during the study. You will be able to eat the foods you usually eat and you will keep your daily protein intake at about 1.5 g of protein per kg body weight. The dietary records will be used to determine your caloric and nutrient intake.

8. You will not increase the amount of time that you already spend doing aerobic training and you will not do any additional weight training during this study.

Training: Training will consist of weight training three times a week for nine weeks. The first week will have only 2 training sessions and week 5 will have 1 training and 1 test session. Each training session will start with a warm-up of 5 minutes of cycling and 5 minutes of stretching. The training exercises will be 4 lower body exercises (incline leg press, parallel squats, leg extension and flexion) and 2 upper body exercises (bench press and lat pull downs, seated row or bicep curl). The training intensity will be 6-8 RM. This intensity is approximately 50-75% of 1 RM. Two sets of each exercise will be performed with 2 ½ minutes rest between sets. A metronome set at 60 beats per minute will be used to help with the timing of the lifting and lowering of the weights. The groups will differ in the time to do the lifting and lowering phases of the exercises. One group will lift the weight in 2 seconds and take 6 seconds to lower it. The other group will do the opposite. All training sessions will be supervised.

Benefits: There is no remuneration, but you will be able to participate in a prescribed and monitored weight training program, and you will be able to meet and exercise with other participants. You will receive your personal strength results for your own information, a summary of the results of the study and a t-shirt. This study will give information related to women and weight training and help with the design of training programs.

Risks: The 1 RM tests will need maximal physical and mental effort. However, the effort that you will need will not be harder than what you have done before in your previous training and testing. The physical training will be based on your individual current maximal strength and will be within your capacity to complete. The study represents little risk to you if you are healthy and actively involved in weight training. A person trained to take blood will take your blood samples. There is a small risk of infection, bruising and small hematoma at the site if not properly cared for. Dr. Tom Martin (M.D.) will do the muscle biopsy procedure under sterile conditions. Two-thirds of individuals feel a "charley horse" sensation of their thigh for a couple of days after the biopsy. Otherwise, complications are infrequent and usually mild. You may feel light headed after the biopsy, and it has been reported that approximately 1 out of 100 will actually faint. This is a temporary condition and results in no long term effects. Bleeding into the muscle or persistent bleeding of the skin occurs 1 out of every 100-250 biopsies. Rare, but more severe complications reported have included puncture of one of the main arteries or nerves of the thigh. These may require surgical repair. These complications have not been seen by the physician performing the biopsy, despite having four years of experience with the technique. No such problems have been seen in this laboratory over the past 9 years. You will report any problems (numbness, excessive pain and swelling) immediately. You will also receive a sheet about the biopsy procedure and care after the procedure.

While serious risk to healthy participants is highly unlikely, they must be known, and you willingly assume the risks related with very hard exercise. There may be unforeseen side effects, and you will be notified if any further risks become known during the study.

Confidentiality: To ensure confidentiality, personal information will be coded and stored in a locked limited access laboratory. Normally, information is retained for a period of five years post publication, after which it will be destroyed. Only the primary investigator will have access to the code sheet and this will be kept in a locked office separate from the data sheets. You will not be identified in any presentation or publication.

Freedom to withdraw: You are free to withdraw at any time without explanation and without any consequences. If you decline to continue or withdraw from the study, your information will be removed from the study upon your request. You will notify one of the investigators if you wish to withdraw.

Additional contacts: If you have concerns about this study, you may contact Dr. Wendy Rodgers, Associate Dean (Research) and Chair of the Faculty Ethics Committee at 492-5910. The Associate Dean has no direct involvement with this project.

Total time commitment: The total time required for all aspects of this study is about 36 hours over 11 weeks.

INFORMED CONSENT

Title of Project: Resistance training with manipulation of concentric and eccentric muscle actions: muscle adaptations.

Principal Investigator: Ellen Gillies, M.Sc. 492-5503
Co-Investigators: Gordon Bell, Ph.D. 492-2018, Ted. Putman, Ph.D. 492-2187, Tom Martin, M.D., Ph.D. 432-0211

Part 2 (to be completed by the research participant):

Do you understand that you have been asked to be in a research study?	Yes	No
Have you read and received a copy of the attached Information Sheet?	Yes	No
Do you understand the benefits and risks involved in taking part in this research study?	Yes	No
Have you had an opportunity to ask questions and discuss this study?	Yes	No
Do you understand that you are free to refuse to participate, or to withdraw from the study at any time, without consequence, and that your information will be withdrawn at your request?	Yes	No
Has the issue of confidentiality been explained to you? Do you understand who will have access to your information?	Yes	No

This study was explained to me by: ______________________________

I agree to take part in this study.

____ ____________________
Signature of Research Participant Date

____
Printed Name

I believe that the person signing this form understands what is involved in the study and voluntarily agrees to participate.

_____________________________________ ___________________
Signature of Investigator or Designee Date

THE INFORMATION SHEET MUST BE ATTACHED TO THIS CONSENT FORM AND A COPY OF BOTH FORMS GIVEN TO THE PARTICIPANT.

Resistance training with manipulation of concentric and eccentric muscle actions: muscle adaptations.

Investigator(s): Ellen Gillies, Ph.D. candidate, Gordon Bell , Ph.D. , Ted Putman, Ph.D., Tom Martin, M.D., Ph.D.

Subject ID ___________

Age _______ years ______months

Menstrual status:

Please indicate which category best fits you now. (Circle the response)

Oral contraceptive use.	Yes	No
Regular periods (consistent and cyclic menstrual cycle lengths of 23-38 days)	Yes	No
Irregular periods (irregular or inconsistent menstrual cycles of 39-90 days)	Yes	No
Infrequent periods (menstrual cycles at intervals of greater than 90 days)	Yes	No

Average length of your cycle ______ days

Start date of your current cycle _______________